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The Sir Richard Stawell Oration.[1]

"DEVILS, DRUGS AND DOCTORS."

By SIR LIONEL WHITBY, C.V.O., M.C., M.A., M.D., F.R.C.P.,
Regius Professor of Physic in the University of Cambridge, England.

SIR RICHARD STAWELL, the very distinguished Melbourne physician, with an outstanding capacity for clinical teaching, was president-elect of the British Medical Association on the occasion of its first and, as yet, only annual meeting in Australia held in 1935. Five months before the meeting Sir Richard developed a coronary thrombosis and died within a few days. This oration, which is given annually by a medical man and a distinguished citizen alternately, was founded in his memory.

As a former President of the British Medical Association I am greatly honoured to be the Richard Stawell Orator for this year. I have chosen as my title "Devils, Drugs and Doctors", partly, I admit, to arouse curiosity, for I might well have called the address "Medicine, Ancient and Modern", or "Prevention is Better than Cure", since I propose to talk mainly about some of the historical aspects of preventive medicine. Each civilization, as it has evolved, has had to provide for medical care and as much prevention against disease as possible.

The manner in which medicine has been practised and the preventive measures which have been taken have obviously depended upon current beliefs and the existing standard of medical knowledge; for example: demonology, in which all illness is attributed to being possessed with a devil—hence the title "devils"; or, a phase of medical practice when illness was thought to be prevented or cured with concoctions, nostrums, herbals, medicines, triturations, infusions and physic—hence the title "drugs"; and lastly, what we believe to be the practice of medicine founded on real and exact knowledge derived from trial and experiment and biological assessment and thereafter practised by an intelligent and highly educated profession. This phase, which began roughly about one hundred years ago, has developed and advanced, especially in these modern days, with geometrically progressive speed. It is because of this important phase in medicine, and the vital position of the men who practise it, that I have completed my title with "doctors".

Prevention is better than cure, and it is profitable to think for a moment of the scope of preventive medicine and of what can in fact be prevented. In general there is hardly any event, or factor, or environment, or everyday incident which may not have an effect on health. This may account for the very large size of our ministries and other departments of health, which are concerned not only

[1] Delivered at a meeting of the Victorian Branch of the British Medical Association on June 5, 1956, at Melbourne.

with health itself but also with such mundane matters as drains, sewers, housing and industry.

The hazards of industry are in fact recognized in a number of picturesque names, such as miner's phthisis, chimney-sweep's cancer, wool-sorter's disease, mule-spinner's cancer, and even weaver's bottom. And these occupational hazards are not confined to industry, for we recognize clergyman's sore throat, student's elbow and boxer's ear.

Much can be prevented on the psychological side, and much illness may have as its foundation an unhappy home life, brutality, nagging, an uncongenial occupation, incompatibility of temperament, jealousy, or an inferiority complex. I mention these aspects of preventive medicine merely to emphasize how broad is my subject.

The successful practice of preventive medicine must depend upon an accurate knowledge of the cause of the condition which is being prevented. Hypothesis leads to proven theory and thus to practical application.

In ancient Egypt the underlying hypothesis for the explanation of all disease was possession with a devil or devils. A well-known papyrus of 1500 B.C. contains a detailed description of the methods for casting out devils, and, even more important, of the ways in which they could be prevented from gaining reentry into the body. Relics of this procedure persisted in medical practice for centuries, even down to relatively modern times, and such stories as that of the Gadarene swine show that the Jews had brought this belief with them out of captivity.

We should respect the demonological view, since in modified form it is practised today by the psychiatrist who, in lengthy sessions, exorcises, temporarily or permanently, the devils of neurosis, instability, unusual behaviour and unhappiness.

A form of medicine perhaps even more ancient than demonology is to be found in practices handed down in folk-lore, which prescribe charms and amulets and other measures for the prevention of disease, as well as natural herbs and animal derivatives for the cure of sickness. Folk-lore forms the basis of "herbalism", which is widely practised today. Almost every town of reasonable size has a herbalist's shop, to which a proportion of the population confidently subscribes. Some of the prescriptions of the herbalist are sound, many of them are extremely artistic, much is sheer charlatanism. But folk-lore medicine, gipsy-craft and so on die very hard, and are quite commonly accepted today by an educated public who are not prepared to neglect any chance of success however strong their faith in orthodox medicine may be.

One might even challenge this present company as to whether there was not one here who had in his pocket something for the cure of rheumatism or for the prevention of a cold—or a cancer—say, a potato, the right forefoot of a hare, a sheep's kneecap, a coffin nail, the skin of a snake or even an iodine locket. Most of such practices are derived from witchcraft medicine and are quite unacceptable to the scientist.

Medicine as practised by the Jews is particularly enlightening. The fundamental belief was that God alone was the healer as well as the one who dispensed disease and disaster to a sinful people. The natural preventive measure was appeasement of the divine wrath with burnt offering and sacrifice. At the same time, the religious rites were conducted by a shrewd and observant priesthood, which had built up a logical code of hygiene and discipline very essential to the health of the people in the days when they were a nomadic tribe.

Leviticus and Deuteronomy can be regarded as the first text-book of hygiene, and Moses was undoubtedly the first medical officer of health. The thirteenth and fourteenth chapters of Leviticus contain precise instructions for dealing with leprosy—the isolation of the patient, the burning of clothes, disinfection, and inspection by a priest.

There are instructions for disinfection in the case of other infectious diseases—and the recognition of the infectious nature of some diseases was a view far in advance of its time—and also for the hygiene of childbirth. The ritual slaughtering of animals, and food inspection, were enlightened hygienic measures, and the prohibition of swine-flesh was possibly a recognition of what we know today—that the flesh of this succulent animal can convey more diseases to man than that of any other animal.

As to field hygiene, the book of Deuteronomy contains precise instructions comparable to those which might be issued to a modern army:

> Thou shalt have a paddle with thy weapon and it shall be, when thou shalt ease thyself abroad thou shalt dig therewith and shalt turn back and cover that which cometh from thee.

The first really sound and orthodox school of medicine was that of Hippocrates in the island of Cos in the Mediterranean, in the fourth century B.C. The teachings of this great school became grafted on to Roman civilization, so that the preventive aspects of medicine as carried out in Rome were in many ways equal to, if not better than, those which exist today. For example, in Rome, in A.D. 100, the population had available 300 gallons of water per head (in London today no more than 35 gallons are available), a water-carriage system for sewage, paved streets, public baths, and markets which were supervised for cleanliness.

Unhappily, with the fall of Rome, and during the subsequent Dark Ages, organized medicine disappeared for about 1000 years, during which time the advances made by the Greeks, the Jews and the Romans passed into oblivion. During this time, in the fourteenth century the world suffered the disaster of the Black Death, which killed more than a quarter of its population, including one-half of the population of London. This and lesser disasters halted the advance of civilization for centuries. It was not until the Renaissance that medicine as we know it, in common with other arts and learning, began to take shape. It was Henry VIII who, on the advice of Linacre and others, founded the Royal College of Physicians in London in 1520, and later, out of the spoils of the monasteries, established a number of chairs of medicine, or physic, in various universities, including the chair which I myself occupy at Cambridge. He has often been called that "munificent donor of other people's properties", but none can deny that his donations were enlightened.

One can gain a vivid insight into the backwardness of the Stuart times by consulting the diary of Samuel Pepys. This great Englishman, in the picturesque, forceful and sometimes coarse language of the seventeenth century, gives in a few brief words a most illuminating sketch of the filth and squalor of London at that time. He says, "This day was much incommoded by the shying of a pot", which accurately describes the sewage disposal system of the times. Even to this day these unbelievably insanitary times are remembered in the battle cry of the medical students of the University of Edinburgh—"Garloo", which is a corruption of *"Prenez garde à l'eau"*.

We can use the Pepys era as a striking example of how readily ignorance can hinder the practice of preventive medicine. This was the time of the Plague of London, from which both Parliament and the Royal College of Physicians took refuge in the country. Deserted London was infested with wild, hungry and starving dogs and cats which were suspected of being the cause of the plague. A benevolent Parliament in retreat ordered their wholesale slaughter. In our present day we know that the main reservoir of plague is the rat, and that the disease is transmitted to man by the bite of the rat flea. Hence, ignorance of cause led to the official slaughter of the natural enemies—dogs and cats—of the primary cause of the pandemic.

In the Pepys era we can also get a picture of the prevention and treatment of disease by "drugs". William Boghurst, a seventeenth century apothecary, lived at St. Giles in the Fields at the time of the plague. He was the author of a book entitled "The Experimental Relation of the Plague", which is one of the best accounts of the times. He made a note of the killing of cats and dogs, and recorded that arsenic amulets had become fashionable

as a prophylactic against the disease. This apothecary stuck to his post and looked after 50 to 60 patients a day, and he had no hesitation in stating in his own advertisement (note, advertisement) in *The Intelligence* of July 31, 1665, referring to his accomplishments: "With wonderful success by God's blessing upon certain excellent medicines which he hath as a water, a lozenge and also an Electuary at but 8d. the ounce price."

Let us look also at a prescription of these times: "Take a chick, or partridge, and three or four larks; take out their entrails and mix with veal knuckles, oranges, lemons, scorzonera roots and (a dozen other queer substances). Boil in a quart of wine and strain. Seven spoonfuls to be taken four times a day."

If we pass now to the eighteenth century, we know that at that time the nation was drunken and illiterate; but it was characterized by a wave of philanthropy showing a foresight far in advance of the squalor of the times. It was in this century that many of our great voluntary hospitals were founded—Guy's Hospital, Saint Thomas's Hospital, the London Hospital, the Middlesex Hospital in London, and even Addenbrookes Hospital in my own University city of Cambridge. It is in this century also that one places the old-fashioned chemist's shop with its colourful display of glass flagons filled with brightly coloured fluids, with its rows of extracts and other ingredients all carefully labelled in Latin in order to preserve the mystery of the apothecary's craft, and having a smell characteristic of the sick room and the hospital. This era, the chemist's shop era, has in part survived to this day. Happily there are still a few old-fashioned shops with coloured bottles; many shops have still, mainly for ornament, shelves filled with Latin-labelled jars, but nowadays the proprietary preparations and tooth-pastes or cosmetics can be sold by anyone in a clean white coat and there is usually not more than one skilled apothecary or pharmacist in any chemist's shop. However, it is as well to remember that the drugs of the eighteenth and nineteenth centuries were in large part useless and quite irrational. Indeed, that charming realist Oliver Wendell Holmes, in the middle of the nineteenth century stated that "if, with the exception of morphia and anæsthesia, the whole materia medica as now used were consigned to the bottom of the sea it would be all the better for mankind and all the worse for the fishes".

The end of the eighteenth century was, however, remarkable for a crucial experiment in preventive medicine, far in advance of the times, from which we draw immense dividends today, and which was a brilliant inspiration arising from simple observation and logical deduction—the guiding principle of scientific medicine as practised today. I refer to the experiment in preventive medicine carried out by Edward Jenner, the Gloucestershire general practitioner, in 1796, which established the cross-immunity between cow-pox and small-pox, and brought into practice, not without opposition even to this day, the technique of vaccination. Jenner's inspiration is said to have come from the chance remark of a dairymaid, who said: "I can't take the small-pox because I have had the cow-pox." This simple country belief and its testing and application by a doctor have controlled a highly mortal and grossly disfiguring disease. It is nowadays difficult to believe that in Jenner's time the beauty of more than 25% of young women was marred in youth by small-pox scarring.

The nineteenth century represents the dawn of exact scientific knowledge and the formulation of rational methods for the prevention and cure of disease based upon an exact knowledge of cause. Without this knowledge, nothing other than chance or experimental observation can lead to success, since one must know one's enemy before he can be conquered. This century from about its mid-point onwards was notable for the development of the sciences of bacteriology, parasitology and immunology, which could not in themselves be explored until the microscope had been sufficiently perfected. The last half of the nineteenth century was distinguished by famous names—Pasteur, Koch, Lister, von Behring, Ross and so on. These were the men who began to control the infective menace at childbirth and surgical operation, who enabled tuberculosis to be studied and prevented, and who paved the way for the elimination and control of malaria—at one time known as million-murdering death. None can read Axel Munthe's "Story of San Michele" with its dramatic description of the first uses of diphtheria antitoxin without being greatly moved.

This was the time, too, when doctors and politicians became aware of the importance of general measures for the preservation of the health of the nation. Hence we have the introduction of factory Acts and other legislation to regulate the hygienic and other standards in places of employment.

The nineteenth century leads naturally into the twentieth in the application and extension of exact knowledge. One thinks of the Plague Commission in 1905, whose findings virtually mean that there will never be another Black Death or even a Plague of London. One remembers the Malta Fever Commission and the rapid development in preventive medicine brought about by the occurrence of three wars within half a century.

Disease under war conditions, often more lethal than destructive weapons, has been greatly controlled. The soldier does not now contract a large number of infectious diseases, or if he does, cure is usually achieved. One thinks of typhoid fever, dysentery, cerebro-spinal fever, tetanus, gas gangrene, cholera, yellow fever, typhus, and the venereal diseases. And should the soldier become wounded, the chance of his survival is some 20 times what it was in the 1914-1918 war, largely owing to the successful control of infection with chemotherapy and antibiotics.

The first half of the nineteenth century will certainly be spoken of as the era in which infection and infectious disease were conquered, and deficiency diseases were recognized and appropriately treated. This century also has been remarkable for the introduction of a large amount of legislation dealing, for example, with the prevention of disease and injuries in industry, and with the control of infectious disease and of cancer, all of which has helped to impel the State to intrude into medicine, since every healthy life is a national asset. Mass radiography and diphtheria and poliomyelitis immunizations are further instances of modern preventive measures.

With this quick and superficial survey of the influence of devils, drugs and doctors in the prevention and cure of disease throughout the centuries, let me now give you a few examples of the impact of disease on history and of how its ravages may alter the face of the world. First, the fall of Rome, which was followed by the Dark Ages and the loss of the culture of all previous civilizations. The fall was undoubtedly due mainly to the political and moral decadence of the nation; but some have brought evidence to show that the decadence may have been in part due to the introduction of malaria into the country by soldiers returning from the campaigns in Asia Minor. Malaria, besides being a disease of considerable mortality, is also the cause of immense morbidity, so that energy is sapped and hard work is impossible. No nation in which malaria is widely spread can be regarded as first-class. Secondly, the Black Death in the fourteenth century. I have already mentioned the mortality. What would be the population of the world now if what we know today had been known then?

And thirdly, in our own times, the 1939-1945 war. What would have been our position in Burma and the Pacific campaigns if we had been unable to control malaria, and at a time when all the supplies of quinine were in Japanese hands? At one time, the casualties in Burma from malaria were some ten times those caused by Japanese weapons of war, and at that time the Director of Medical Services was a more important officer than the first officer of the General Staff. The medical services in that campaign eventually reduced the malarial casualties to no more than the number of wounded.

Civilization brings penalties as well as benefits. With each advance in science, or in amenities, or in transport, new problems arise. The human body was not designed to

be hurtled through the air in excess of the speed of sound. From television emanate small but appreciable amounts of ionizing radiations, and also from wrist watches and X-ray plants used in the fitting of shoes. Preventive medicine has, indeed, never finished with the problem of preventing.

And though indeed we may be proud of the advances that have been made in the past century—which I have called the era of doctors—we must never fail to realize how limited these advances are, and how much more, infinitely more difficult, there is to be done. We have to a large extent gained control of many factors and forces extraneous to the human body; but diseases which are inherent in man himself, such as rheumatism, arterial disease and cancer, have yet to be conquered. Medicine itself is never a closed book. The story is never finished. Not infrequently the control or eradication of one disease reveals or seems to produce another. All this, in fact, makes up the fascination of the practice of medicine. Drama is always just around the corner, whether it be the drama of discovery, the drama of cure or even the inevitable final drama. In all such dramatic episodes, even to this very day, you will almost inevitably find doctors as well as drugs, whilst Satan, the Devil himself, seems also quite often to be concerned.

ANTE-NATAL CARE AND THE PREVENTION OF ECLAMPSIA AND SEVERE TOXÆMIA.[1]

By T. DIXON HUGHES,
Sydney.

IN April, 1951, at the first Australian conference of Fellows and Members of the Royal College of Obstetricians and Gynæcologists, I read a paper showing the incidence of eclampsia in 15,000 booked cases as one in 3000 from 1948 to April, 1951 (Hughes, 1951). It is now proposed to review results up to June, 1955. There are now 29,681 booked patients who were delivered in the hospital during the whole period. To avoid any misunderstanding the word "booked" means any patient who has attended the out-patient department at least once before being admitted to hospital. Table I shows the number of booked patients delivered and the incidence of eclampsia.

TABLE I.

Period.	Number of Booked Patients Delivered.	Number of Cases of Eclampsia.
1948–1949	4532	2
1949–1950	4512	3
1950–1951	4379	0
1951–1952	3951	1
1952–1953	4318	0
1953–1954	4134	3[1]
1954–1955	3855	0

[1] One of these three patients had one convulsive seizure, and another seizure fourteen days later when apparently well.

The incidence is one in 3297 over the total time since June, 1948. During the last five years, in a series of 20,637 patients delivered there were four eclamptics, including the problematical one, which gives an incidence of one in 5000. Therefore we have been able to maintain and improve our low incidence, and at one stage we had 13,000 consecutive patients with the occurrence of one case of eclamapsia. I think you will agree that these figures are too large and that they cover too long a period to be attributed to mere chance. The question arises, by what means are these results obtained? It is now proposed to discuss these in more detail.

1. Above all, the whole staff must be keen, from the junior nurse in the out-patient department to the senior obstetrician. They must be eternally vigilant for the early warning signs of toxæmia, which Sir Bernard Dawson (1953) aptly states as being often disregarded because of their apparent insignificance.

2. There must be an educated population—one that appreciates the results of adequate ante-natal care, so that mothers are prepared to carry out ordinary commonsense hygiene and cooperate by regular attendance at the clinic. This implies a reasonable standard of living; but Nelson (1955) shows that eclampsia is not peculiar to the rich or to the poor.

3. The blood pressure must be properly evaluated. This is of paramount importance. Unless the significance of the individuality of blood pressure is appreciated, adequate ante-natal care is not being carried out.

Blood Pressure.

The appreciation early in my career of the individuality of blood pressure probably played a major part in allowing me to pass through my active obstetric years with over 5000 deliveries without the occurrence of a case of eclampsia, and the adoption of this principle of the individuality of blood pressure at the Women's Hospital, Crown Street, is represented in the results that have been achieved. It is to be hoped that all reference to any set standard of height of blood pressure will be omitted from all discussions on the subject, and that the discussion will depend upon the number of millimetres of mercury by which the pressure has risen from the patient's own standard. In other words, we adopt the principle of the relative rise of the blood pressure—that is, the pressure under discussion related to the patient's original blood pressure. The importance of this has been expressed in a previous paper (1951). Let me, at this stage, quote M. Hamilton, G. W. Pickering, J. S. Fraser Roberts and G. S. C. Sowry, of the Department of Medicine, Saint Mary's Hospital, London (1954), who state that "obviously since there is no sharp dividing line between clearly normal and clearly abnormal levels of blood pressure, even a statistical definition must be arbitrary". They further state, in discussing hypertension, that "the harm comes when we draw a rigid line, and say that above this line the pressure is abnormal, below it normal". And in their conclusions they state that "the data presented in this paper have led us to doubt whether there is any justification, other than that of convenience, for drawing a line dividing normal from abnormal pressure". Therefore it is appropriate at this stage to state the standards in respect to blood pressure that are employed at our hospital and standardized in the out-patient department. They are as follows: (i) A rise of five millimetres of mercury in the diastolic blood pressure means that the patient must be observed within seven days after the twentieth week. (ii) If the diastolic pressure has again risen within the seven days, the patient must be examined within at least two or three days according to the magnitude of the rise, or admitted to hospital if any other sign of toxæmia has developed. (iii) If weight increase is associated with a relative rise in the diastolic blood pressure irrespective of successful weight control, that patient must be examined weekly.

This last instruction is inserted so that the attendant physician will not be led astray by the fact that he has reduced the hidden œdema, as shown by the weight loss, and disregard the more dangerous sign, to my mind, that the blood pressure has not returned to the patient's normal reading. Moreover, it can be taken as axiomatic that if there is a relative rise of blood pressure associated with an excessive weight gain, that patient has toxæmia, and she must be ever suspected of being likely to present a deterioration in her condition which may ultimately be rapid. The question of weight increase will be dealt with

[1] Read at a meeting of the Section of Obstetrics and Gynæcology, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

later; but sufficient has been said to show the importance that is attached to the relative rise in diastolic pressure at the Women's Hospital, Crown Street.

Weight Gain.

Hamlin (1952), Stevenson (1952) and Nelson (1955) have discussed weight gain at some length. Hamlin (1952) believes that any woman who gains more than eight pounds from the twentieth to the thirtieth week runs a considerable risk of developing preeclampsia. Stevenson (1952) states that women who are overweight at the beginning of pregnancy are the most prone to develop eclampsia. Nelson (1955), in a detailed study, states that "there is a tendency for those who later develop pre eclampsia to gain excessively in the twenty to thirty week period. Nevertheless more than half the normal cases gained excessively, and one quarter of the cases of pre eclampsia toxæmia were in the lesser gain group". However, most figures show that at least 75% of patients who develop severe toxæmia show excess gain of weight.

The policy of the Women's Hospital is clearly defined in its instructions to clinical assistants and others; it states that after twenty weeks a weight increase of over one pound per week must be checked within seven days, and as was previously mentioned, that if the weight increase is associated with a relative rise in blood pressure, irrespective of successful weight control, the patient must be examined weekly. But it is appreciated that certain patients may develop severe toxæmia and yet fail to show any appreciable weight gain, and so the absence of a gain in weight does not exclude a possibility that toxæmia or eclampsia may develop. In the days of eclampsia I personally dreaded the admission to hospital of a non-œdematous patient having convulsions, who was termed "a dry eclamptic" for want of a better term. This is borne out by a report of the Committee of the Section of Obstetrics and Gynæcology of the Royal Society of Medicine (1922); amongst criteria given as signs of bad prognosis was absence of œdema, which was associated with a mortality rate of 30%, as against 15% when œdema was present. Nevertheless the literature does show that the greater majority of patients who develop toxæmia signify their intention of doing so by a sudden gain in weight; the closer to term, the more certain it is to herald the onset of toxæmia. To neglect such a sign is to give inadequate ante-natal care. The statistics presented by Nelson (1955) do not justify his statement that "the relationship of weight gain to pre eclampsia, and in particular the severe grade, is too indefinite to be of real practical value in predicting the development of pre eclampsia; and dietary control is, in fact, unlikely to benefit those who require help most". It must be borne in mind that those statistics are based on the weight gain only between the twentieth and the thirtieth weeks, with the elimination of the last ten weeks of pregnancy, when a sudden gain will give that warning for closer observation. Hamlin (1952) was searching for an earlier sign of preeclampsia. That this can be found in 75% of cases means that it is a sign worth looking for. But it is readily accepted at this hospital that patients may exceed that limit without the development of toxæmia, and 25% who show no gain in weight may later develop toxæmia. As was mentioned before, no definite instructions are given except that a woman who gains more than one pound per week must be examined within seven days. The purpose of this is to endeavour to discover the possible cause of the increase in weight. Is it associated with any other sign such as œdema? Is it dietary excess? Is the patient taking an ill-balanced diet? There are few women who appreciate gaining half a stone a month, and most will gladly accept advice from the dietitian. But it is understood that to rely on any one sign as infallible is to court disaster. However, any gain in weight, particularly if sudden, is worthy of attention, and to decry the need for regular observation of weight is a retrograde step.

Diet.

The diet advised by the hospital is much the same as that laid down by the Department of Maternal and Baby Welfare of New South Wales and based on the findings of the Nutrition Committee, National Health and Medical Research Council of Australia (1954). It is supervised by the dietitian, who gives the mothers lectures on food values, and on the advantages of a well-balanced diet of approximately 2200 Calories. The mothers are advised to have approximately 85 grammes of protein in the form of milk, meat, fish, cheese and eggs; but in all probability this figure is not attained by a fair proportion of patients. Warning is given against ingestion of excessive fat and carbohydrates in the form of cakes, sweets and excessive bread and butter, which interferes with their having their properly balanced diet. Salt restriction is advised from the fifth month onwards. In the event of any patient gaining excessive weight after a clinical assessment, she is interviewed by the dietitian, who checks on her diet and perhaps finds some gross error. On the other hand, some patients have been underweight, and with improved nutrition they do exceed for a while the arbitrary gain of one pound per week. However, an attempt is made to assess these individually. However, there are the patients with hidden œdema; these are greatly benefited by early control of sodium intake, and above all by care in seeing that they are not suffering from hypoproteinæmia as a result of restriction of protein. I myself have found that when sodium retention is suspected, an almost salt-free diet may be obtained from a diet of two eggs, one and a half to two pints of milk, as much fresh fruit as the patient wishes, and no other food. This is taken for twenty-four hours, in combination with as much rest as possible. The result is frequently dramatic; the patient settles down, and provided careful watch is kept, she may reach term perhaps with one or two repetitions of the milk and fruit regime. The essential point is common sense in the diet and the assessment of the individual patient. There are too many authenticated references to show the beneficial effect of sodium chloride restriction to allow its value to be doubted, and in this regard it would appear unnecessary to advise doctors to refrain from giving their pregnant patients antacid compounds containing sodium. A few months ago a patient returned with an instruction sheet, amongst the instructions being the advice that if she developed heartburn she was to take soda mints or "Bisodol". So this pitfall is again brought to notice. It is not the purpose of this paper to discuss the undoubted advantage of sodium restriction and its elimination.

Albuminuria.

The finding of albumin in a catheter specimen of urine is looked upon as a serious sign, and the patient is recommended for admission to hospital for the determination of its cause. Sir Bernard Dawson of New Zealand (1953) endorses this statement, and goes even further when he states that "among these prodromata of the final catastrophe of eclampsia we include albuminuria, regarding proteinuria as a late and dangerous sign and believing that toxæmia of pregnancy can be recognized and treated prior to its appearance".

The Role of Administration.

There are two aspects of administration that are worthy of attention. One is to check that patients are being examined at the required intervals as laid down in the hospital procedure. In the stress of out-patient work and changing personnel, through illness and holidays, often a slight deterioration in patients can be overlooked, and the patient may be allowed too long a lapse of time before she is requested to return. To avoid such a mishap, all the cards are checked at the end of each day to see that no rule has been overlooked, and if necessary contact is made with the patient and she is asked to return at an earlier date.

On the out-patient's card are two columns, in one of which is noted the number of days after which the patient is to return. The other column is for the initials of the attending officer who gave the instructions. This is of help in placing responsibility on an individual. The cards are then filed, a separate record is made of the date for their return and they are checked on that date.

TABLE II.

Place.	Severe Toxæmia.	Mild Toxæmia.	Place.	Severe Toxæmia.	Mild Toxæmia.
Aberdeen, 1943–1945	3·0%	15·4%	Aberdeen, 1951–1953	6·8%	16·6%
The Women's Hospital, 1947 ..	5·6%	5·5%	The Women's Hospital, 1954 ..	3·2%	11·5%

Any patient who fails to attend is either sent a letter or, if she is in a late stage or has shown signs of deterioration, more active measures are taken, such as arranging for her to be visited. If a patient fails to report, she is notified three times that unless she will cooperate with the hospital regulations her booking will be cancelled. This rarely has to be carried out. It is thought that the work put into checking the cards by the out-patient registrar has been of immense help in reducing those errors that can and do occur unless an ever-vigilant watch is kept. Some of the patients who live in the outer suburbs are very ably cared for by clinics supervised by the Department of Public Health; but these clinics care for the patients according to the standards of our hospital, and refer them back to the out-patient department if there is any deviation from the normal in any respect.

Number of Ante-Natal Visits.

In view of the foregoing statement, it may be asked what are the rules governing the attendance at the out-patient department. It can be summarized in the statement "as often as the condition of an individual patient warrants it", and any other system must fail. The usual procedure is for the patient to be examined monthly up to the twenty-eighth week unless any complication arises, then perhaps fortnightly to the thirty-sixth week if no sign of overweight or any abnormality has arisen, and then at least weekly to term. The average number of visits by patients who reported to the hospital at eight to twelve weeks was 15; the minimum was 13. Visits frequently exceed 15. This may appear to be excessive by old standards, but is repaid by the reduction of the incidence of eclampsia and severe toxæmia.

Relationship of the Incidence of Severe Toxæmie to More Intensive Ante-Natal Care.

The experience at the Women's Hospital is directly opposite to that reported by Nelson at the Aberdeen Hospital (1955), which prompted the statement that "severe pre eclampsia has increased despite the improvement in antenatal care". Table II shows that at the Women's Hospital there are approximately the same number of cases of toxæmia in both years reviewed. The proportion of severe toxæmia is 50% of the toxæmias for the period 1947-1948, whereas for the period 1953-1954 it is 21%. In reality the results are better than the figures show, as the degree of albuminuria in the earlier year was much more severe than that in the latter period. The standard laid down by Nelson (1955) was adopted for comparison purposes only, and *primiparæ* only are discussed. Briefly, the standard set was as follows: mild when the diastolic blood pressure is 90 millimetres of mercury or more and albumin is absent; severe when albuminuria is added to the foregoing (a trace of albumin in a non-catheter specimen of urine is disregarded).

Our experience has been borne out by that in New Zealand, as shown by Sir Bernard Dawson in Dunedin (1953), and by the magnificent results reported by Corkill (1952), showing that for the whole of the Dominion of New Zealand the incidence has been reduced from 3·07 per 1000 births to 1·4 per 1000 births.

The Relationship of Premature Neonatal Death Rate to the Improvement in Ante-Natal Care.

That the premature neonatal death rate falls as a result of improved ante-natal care is best demonstrated by the fact that when the incidence of eclampsia in 1947 was one in 400, the premature neonatal death rate was 15·6%. Over the last five years the incidence of eclampsia has been one in 5000 and the average premature neonatal death rate for that period has been 10·3%, and for the last twelve months 8·8%. One of the factors influencing this is the fact that whereas 50% of the premature babies previously born weighed under five pounds, now more than 50% of the premature babies weigh over five pounds.

The Use of Cæsarean Section.

Meares in another paper will show that in the years from 1950 to 1954 hypertension toxæmia was the sole indication for Cæsarean section in only four booked patients.

Summary.

At the risk of repetition, I would summarize our procedure by restating the following essentials:

1. Ante-natal care must be adequate and must comply with a high standard of efficiency, demanding an exacting control of patients almost personal in nature.

2. The blood pressure must be properly assessed in relation to the basal pressure.

3. The weight must be carefully observed, and its significance as an indication for treatment, control and closer observation must be appreciated.

4. Previous arbitrary ideas of how often a patient should be examined must be abandoned.

5. It must be recognized that the presence of albumin in a catheter specimen of urine is a late sign of toxæmia, and that control must commence before its appearance.

Acknowledgements.

I should like to thank Professor F. J. Browne and Professor B. Mayes for their encouragement and for their faith in adequate ante-natal care. To members of the team at the Women's Hospital—honorary obstetricians, superintendent, resident medical officers and the nursing staff—who have made these results possible, I offer my sincere thanks.

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Addendum.

During the year beginning July 1, 1955, no case of eclampsia has occurred among booked patients at the Women's Hospital, Crown Street. There have thus been two successive years with no eclampsia among booked patients.

INDICATIONS FOR CÆSAREAN SECTION: A CONSIDERATION OF CERTAIN ASPECTS.[1]

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DELIVERY of a baby by Cæsarean section is an alternative to vaginal delivery. This being so, the obstetrician uses his judgement to make the choice. The judgement is based on knowledge and experience, the experience of others modified by one's own. It is in the exercise of this choice that the wisdom of the obstetrician is tested to the full.

In "The Year Book of Obstetrics and Gynecology 1954-1955", Greenhill comments that every hospital should have a committee to review the chart of every patient who undergoes Cæsarean section. By this means Shir and associates (1953) at the Brooklyn Jewish Hospital in a period of four years reduced the incidence of Cæsarean section by almost 25%.

With the idea that it is better to shut the stable door before the horse has bolted, for many years at the Women's Hospital, Crown Street, there has been observed a rule that before a junior obstetrician (several of whom have had over twenty years' obstetrical experience) decides on delivery by Cæsarean section he must consult with a senior. There is no doubt that the ready adherence to this practice has led to careful and wise consideration before the operation is performed.

Drew-Smythe (1950) has made the following statement:

> It is the supervision of the pregnancy, control of labour and the recognition of the case in which Cæsarean is the best line of treatment that ensure a live mother and a live child.

TABLE I.

Years.	Incidence of Eclampsia.	Incidence of Cæsarean Sections for Preeclamptic Toxæmia.
1946 to 1950 ..	1 in 1249	1 in 677
1950 to 1954 ..	1 in 4210	1 in 4210

Prophylaxis.

What improved ante-natal supervision and care can do in decreasing the incidence of Cæsarean section is shown in Table I. The figures refer to booked patients at the Women's Hospital, Crown Street, and two four-year periods are compared.

Thus a threefold decrease in the incidence of eclampsia led to a sixfold decrease in the number of Cæsarean sections for preeclamptic toxæmia.

That good ante-natal care will improve the results from Cæsarean section has been shown by Dunlop (1954), of Edinburgh (Table II).

The stillbirth rate in this series was almost three times as high for unbooked as for booked patients.

It is undoubted that better use of trial labour will decrease the incidence of Cæsarean section. Too frequently do we hear the history that in the first pregnancy a Cæsarean section was performed for disproportion without a trial of labour. Many of the patients with this history have come from abroad.

The Choice.

Before the various conditions which sway our choice are discussed, it is necessary to consider the safety or otherwise of the operation to the mother and baby, and what sequelæ there may be.

[1] Read at a meeting of the Section of Obstetrics and Gynæcology, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

Cæsarean section is considered only when pregnancy or labour has ceased to be physiological. Therefore any mortality or morbidity is that of the pathological state added to those of the operation itself.

During the five-year period, from July 1, 1949, to June 30, 1954, 22,684 patients were delivered of 22,981 infants at the Women's Hospital, Crown Street. Of these, 6·2% were unbooked and 52·5% were primiparous. The maternal mortality rate per 1000 live births and stillbirths was 0·4,

TABLE II.

Edinburgh Series, 1940–1949: Cæsarean Section.

Patients.	Number of Patients.	Stillbirth and Neonatal Death Rate.	Stillbirth Rate.
Booked ..	1200	9·0%	2·9%
Unbooked ..	400	21·0%	8·0%

and the perinatal death rate (combined stillbirth and neonatal death rate) per 1000 live births was 31·0. A total of 327 Cæsarean sections, 59 (16·7%) of the patients not being booked, was performed without a maternal death.

For the previous five years (1944-1949) the Cæsarean section rate averaged 1·5%. During the five years from 1949 to 1954 it fell from 1·9% to 1·0% (Table III). (The

TABLE III.

Cæsarean Section Rate (Percentage).

Year.	Cæsarean Section Rate.
Average, 1940–1954 ..	1·4
1949–1950	1·9
1950–1951	1·8
1951–1952	1·2
1952–1953	1·0
1953–1954	1·0

figures in Table III refer to all public patients, booked and unbooked, at the Women's Hospital, Crown Street, Sydney.)

A more comprehensive picture is obtained by grouping the rates from the four University obstetrical teaching hospitals in Sydney (Table IV).

Maternal Mortality Rate.

At the Women's Hospital, Crown Street, there were no maternal deaths in this series of 327 Cæsarean sections for all indications. In fact, it is over six years since a death has followed Cæsarean section; that is, no death has followed over 376 operations.

In the Edinburgh series (1954) for the ten years 1940 to 1949 inclusive, 1796 Cæsarean sections were performed. The maternal mortality rate was 1·8% for the first five years and 0·5% for the last. Dunlop remarks that:

> . . . the figure for maternal mortality in a Cæsarean series is now so satisfactorily low that it has ceased to be an index by which to judge the relative success or failure of different techniques and procedures.

With this I agree, and later I shall discuss the problem of the indications for Cæsarean section with emphasis on the fœtal point of view.

Nevertheless, let not these results make us over-confident, for in the Metropolitan Health District of Sydney in the period from 1944 to the present there were (if Cæsarean section for rupture of the uterus is excluded) 49 deaths following Cæsarean sections.

Maternal Morbidity Rate.

The notifiable pyrexial rate *per centum* is taken as the guide to the morbidity rate. In Edinburgh, notifiable pyrexia is a temperature of 100·4° F. repeated during

TABLE IV.

Cæsarean Section Rate, Period 1949 to 1954.

Hospital.[1]	Average for Five Years.	1949–1950.	1950–1951.	1951–1952.	1952–1953.	1953–1954.	Perinatal Mortality Rate.
King George V Hospital	3·02%	2·1%	2·9%	2·7%	3·1%	4·3%	9·8%
Royal Hospital for Women	1·74%	1·8%	1·7%	1·8%	1·3%	2·1%	10·7%
Royal North Shore Hospital	2·66%	2·3%	2·6%	2·6%	2·6%	3·2%	9·6%
Women's Hospital, Crown Street ..	1·38%	1·9%	1·8%	1·2%	1·0%	1·0%	8·5%

[1] The figures were supplied by the medical superintendents. It should be noted that a higher Cæsarean section rate is not accompanied by a lower perinatal death rate.

twenty-four hours or sustained during that time, occurring in the first twenty-one days of the puerperium. In New South Wales a temperature of 100·4° F. or higher must occur on more than one day in the first ten days of the puerperium. In the Edinburgh series the rate was 13%, and in the Crown Street series 15%.

Perinatal Death Rate (Combined Stillbirth and Neonatal Death Rate).

Let us now look at the problem from the fœtal point of view. Some figures from abroad are set out in Table V.

TABLE V.

Country.	Source.	Years.	Number of Cæsarean Sections.	Perinatal Mortality Rate.
United States	Litchfield *et alii*	1945–1951	969	4·5%
	King *et alii* ..	1947–1952	1219	5·76%
	Little *et alii* ..	*circa* 1951	(*a*) 225	6·2%
			(*b*) 179	3·4%[1]
Great Britain	Dunlop *et alii*	1940–1949	1818	11·1%

[1] After introduction of intratracheal and gastric suction after delivery.

The perinatal death rates associated with Cæsarean section for the four University teaching hospitals in Sydney are set out in Table VI.

At the Women's Hospital, Melbourne, for the two years 1952 to 1954 the average perinatal death rate was 11·2%.

J. W. Johnstone (1951) found that in 1718 Cæsarean sections in the thirty-year period from 1919 to 1949 at the Women's Hospital, Melbourne, the perinatal death rate was 15·4%.

King *et alii* (1953) state that in a series of 1003 Cæsarean sections (performed mostly under spinal anæsthesia), in which the fœtal prognosis was excellent, there were 11 neonatal deaths. Thus the perinatal mortality rate from the operation itself was at least 1%.

If patients with *placenta prævia* are excluded from the 1949-1954 series at the Women's Hospital, Crown Street, the perinatal death rate following Cæsarean section is 4·3%—that is, the rate is halved.

Remote Effects of Cæsarean Section.

Chesterman (1953) found a decrease in fecundity equivalent to a loss of one baby to the community with every two Cæsarean sections performed. This decrease was mainly due to "repeat" sections, for of women not sterilized at the second Cæsarean section only one in three had a third pregnancy.

The knowledge that she has not been able to have a baby *per vaginam* may give a mother a feeling of inferiority, and make her fearful of becoming pregnant and anxious when she is. The presence of the scar and the fact that it may rupture (1·76%—Chesterman, 1953) mean that the mother for safety must have her confinement in a large centre of population, and must choose a well-equipped hospital, be under close supervision and ideally in hospital in the last weeks of the pregnancy. The obstetrician asked to undertake her care has the ever-present anxiety that the scar may rupture during the pregnancy or labour. The presence of the scar may modify the treatment of some other complication of pregnancy.

If the reason for performing the first Cæsarean section was a recurring one, such as gross pelvic contraction, the obstetrician is saved the responsibility of a subsequent labour. It is when it has been performed for a non-recurring indication that the judgement of the obstetrician is fully taxed.

Before embarking on a Cæsarean delivery, one must look further than the immediate problem of the present pregnancy to the whole obstetrical future of the mother. Chesterman has shown that this is especially so when a second Cæsarean section is being contemplated.

Let us now summarize the results of Cæsarean section on mother and child (Table VII).

It is against this background that we must make the decision whether or not to perform a Cæsarean section.

Indications for Cæsarean Section.

Let us study the states which caused the obstetrician to choose Cæsarean section in the Crown Street series, 1949 to 1954. Only the first listed indication is considered. Alongside these figures we shall put those of the Edinburgh series, 1940 to 1949 (Table VIII). However, it would be wrong to compare the two series except in a general way.

It is clear that the indications mechanical dystocia, uterine scar, hæmorrhage and hypertensive toxæmia make up over 70% of the indications for Cæsarean section in each of the series.

As in the last four years, 1950 to 1954, at Crown Street, only eight Cæsarean sections (3·4%) have been performed for hypertensive toxæmia in both booked and unbooked cases, it has now become an infrequent indication for Cæsarean section. In fact, during the last four years only four Cæsarean sections have been performed on booked patients with hypertensive toxæmia as the prime indication. This being so, I shall now confine the discussion to

TABLE VI.

Perinatal Death Rates Associated with Cæsarean Section.

Hospital.	Average for Five Years.	1949–1950.	1950–1951.	1951–1952.	1952–1953.	1953–1954.
King George V Hospital	9·8%	8·0%	10·8%	10·6%	8·9%	10·6%
Royal Hospital for Women	10·7%	22·4%	12·1%	11·7%	6·1%	1·5%
Royal North Shore Hospital	9·6%	10·0%	8·0%	4·0%	18·0%	8·0%
Women's Hospital, Crown Street	8·5%	6·5%	10·1%	9·6%	8·3%	8·7%

the three common indications, accounting for over two-thirds of the Cæsarean sections at Crown Street—namely, mechanical dystocia, uterine scar and hæmorrhage.

Mechanical Dystocia.

"A live birth may be accomplished through a pelvis which possesses a clear available space of three inches in the conjugate." (Ramsbotham, 1841.) At that time Cæsarean section was often a lethal operation.

At Crown Street, for many years every *primipara* with a normal presentation and a true conjugate of more than

TABLE VII.

Results of Cæsarean Section for All Indications.

Subject.	Rate *per Centum*.	Source of Figures.
Mother:		
Mortality	0·5	Edinburgh Series, 1940–1949.
Morbidity	15	Women's Hospital, Crown Street.
Rupture of scar in subsequent pregnancy	1·76	Chesterman (1953).
Fœtus and infant:		
Perinatal mortality	8·5	Women's Hospital, Crown Street.
Perinatal mortality for the operation itself	1	King *et alii* (1953).

3·5 inches (8·4 centimetres) has been given a trial of labour if disproportion was suspected.

Elective Cæsarean section may be performed when the disproportion is extreme, or when it is associated with (i) a malpresentation which cannot be corrected, (ii) advanced age of a *primipara* (over forty years), (iii) a bad obstetrical history, (iv) a scar on the uterus, (v) many years of sterility, (vi) a *placenta prævia*. In addition, the great importance of a first baby to an Rh-negative mother must be borne in mind.

TABLE VIII.

Indication for Cæsarean Section: Booked and Unbooked Patients; Primary and Repeated Sections.

Grouped Indications.	The Women's Hospital, Crown Street, 1949 to 1954: 327 Cases.	Edinburgh Hospitals, 1940 to 1949: 1796 Cases.
Mechanical dystocia[1]	27%	39%
Uterine scar	22%	4%
Hæmorrhage	19%	11%
Hypertensive toxæmia	6%	17%
Incoordinate uterine action	3%	12%
Maternal disease	2%	6%
Tumours	2%	2%
Miscellaneous and others	62 cases	162 cases

[1] Malpresentation is included under the heading "mechanical dystocia".

Of the 89 patients who had a Cæsarean section performed for mechanical dystocia (which included malpresentation—for example, transverse lie) in the years 1949 to 1954, 10 had previously undergone a Cæsarean section, and in 15 cases it would not have been wise for a trial to be given (malpresentation eight, previous stillbirth or neonatal death three, fœtal distress two, sterility one, contracted outlet one). Of the 64 other patients, 36 were given a trial of labour, 31 for longer than six hours, 18 for more than twelve hours, nine for more than twenty-four hours and four for more than forty-eight hours.

The trials of labour have been managed along the general lines set out by Hawkesworth (1952) in his excellent address to the Royal Society of Medicine, London.

In the four years 1950 to 1954, 143 patients were given a trial of labour. These 143 cases are set out alongside Hawkesworth's series of 124 cases in Table IX.

These figures, as all others in this paper, and those of Hawkesworth, are uncorrected.

It is seen that in both series more patients whose trial of labour lasted over twenty-four hours were delivered vaginally than by Cæsarean section (Cæsarean section rate 26·5% in the Crown Street series). As Hawkesworth observes, "therein lies justification of the trial".

The causes of the six perinatal deaths in the Crown Street series were as follows: congenital heart abnormality, two; encephalocele, one; intracranial hæmorrhage, one; pulmonary hæmorrhage, one; cerebral hæmorrhage following an eclamptic convulsive seizure, one.

Prolonged Labour.

From the fœtal point of view, a prolonged labour from any cause is to be feared. This was shown by Dixon

TABLE IXA.

Trial of Labour: Oxford Area Department, 1948–1951, and The Women's Hospital, Crown Street, 1950–1954.

Place.	Total Deliveries.	Number of Trials.	Maternal Mortality.	Cæsarean Section Rate.	Perinatal Mortality Rate.
Oxford	10,686	124 (1·1%)	0	41·1%	1·6%
Women's Hospital, Crown Street	18,032	143 (0·7%)	0	32·9%	4·5%

Hughes (1941) to be true in cases of posterior position of the occiput. Among *primiparæ* delivered of a live baby, labour lasted for an average of thirty hours; among those whose babies were stillborn or died from causes attributable to the posterior position of the occiput, the average duration of labour was fifty-four hours. That it is so from

TABLE IXB.

Trial of Labour over Twenty-four Hours.

Place.	Number.	Number of Cæsarean Sections.	Cæsarean Section Rate.
Oxford	73	31	42·0%
Women's Hospital, Crown Street	49	13	26·5%

all causes is shown in Table X. The figures in this table refer to 269 prolonged labours (lasting over forty-eight hours) at the Women's Hospital, Crown Street, for the period from 1949 to 1954.

TABLE X.

Results in 269 Cases of Prolonged Labour, 1949 to 1954.

Method of Delivery.	Number of Cases.	Perinatal Deaths.
Cæsarean section	12	0
Vaginal delivery	257	26 (10%)

In prolonged labour vaginal delivery resulted in a 10% perinatal death rate. Undoubtedly there is some neonatal morbidity to be added also, though I cannot present figures for this.

Uterine Scar.

One of the striking differences in the frequency of the various indications in the Edinburgh series (1940-1949) and the Women's Hospital, Crown Street, series (1949-1954) is the much greater frequency of uterine scar as the prime indication in the latter series. At Crown Street this

incidence has risen from 8% in the five years 1944 to 1949 to 22% in a similar period 1949 to 1954—almost treble in five years.

The term "uterine scar" for practical purposes refers to the scar from a previous Cæsarean section. In the Edinburgh series, in which 74 scars were the indication for Cæsarean section, only five did not result from a previous Cæsarean section, and in the Crown Street series, of 73 scars, only two did not result from a previous Cæsarean section.

In the Crown Street series of 327 patients delivered by Cæsarean section, 100 (31%) had undergone a previous Cæsarean section. At the Women's Hospital, Melbourne, in the two years from 1952 to 1954, 433 Cæsarean sections were performed, 146 (33%) being "repeat" operations.

Thus in one-third of the cases in which the choice fell to Cæsarean section, the judgement had been previously biased.

In the four-year period 1950 to 1954, at the Women's Hospital, Crown Street, there were 194 patients who had previously been delivered by Cæsarean section. Of these, 15 had already undergone more than one Cæsarean section. The risk of vaginal delivery was not taken in these 15 cases.

Thus Cæsarean section had been performed only once on each of 179 patients.

In only 76 of these cases was the Cæsarean section repeated. That is, 67·5% (more than two-thirds) of these patients were delivered vaginally in a subsequent pregnancy after having had one previous Cæsarean section.

If Cæsarean section had been performed on all these patients, on the principle of "once a Cæsarean, always a Cæsarean", the Cæsarean section rate would have risen from 1·4% to 2·1%. That is to say that instead of 200 Cæsarean sections, 300 would have been performed.

In Table XI are set out the methods of delivery of 179 patients at the Women's Hospital, Crown Street, who had had only one previous Cæsarean section.

TABLE XI.

Patients Who had had Only One Previous Cæsarean Section.

Method of Delivery.	Number of Patients.
Vaginal	103 (67·5%)
Repeat Cæsarean section	76 (32·5%)

Table XII shows the number of perinatal deaths of infants whose mothers had had one or more previous Cæsarean sections at the Women's Hospital, Crown Street, in the period from 1950 to 1954.

TABLE XII.

Perinatal Deaths After One or More Cæsarean Sections.

Method of Delivery.	Number of Cases.	Number of Perinatal Deaths.	Perinatal Death Rate.
Vaginal delivery ..	103	12	11·6%
Repeat Cæsarean section	91	6	6·6%

From this it would appear that after a previous Cæsarean section the safer mode of delivery for the infant is by another Cæsarean section.

In Table XIII are shown the causes of perinatal deaths of infants whose mothers had had one or more Cæsarean sections at the Women's Hospital, Crown Street, in the period from 1950 to 1954.

When the cause of the death is analysed, the difference is hardly significant.

One scar ruptured amongst the 194 patients (1950-1954). This patient was not being given a trial of labour. The scar ruptured at the onset of labour. It was from a classical Cæsarean section.

Of the 76 patients delivered by Cæsarean section, some had a recurring indication (for example, 15 had mechanical dystocia in the first labour) which might rule out a trial of labour in a subsequent pregnancy. Twenty-nine of the 76 (38%) were not given a trial of labour.

Of the remaining 47 (62%), only 16 (34%) were given a trial longer than six hours.

In view of the absence of rupture of the scar in this series, if the 31 representing almost half of the 76 delivered by repeated Cæsarean section had been given a longer trial of labour, possibly the Cæsarean section rate would have been further reduced without undue increase in the risks to mother and baby. This is worth further consideration, in view of Chesterman's finding on the decrease in fecundity after the second Cæsarean section.

TABLE XIII.

Causes of Perinatal Deaths After One or More Cæsarean Sections.

Method of Delivery.	Maceration of Fœtus.	Congenital Heart Abnormality.	Prematurity.	Cause Unstated.
Vaginal delivery	2	1	6	3
Repeat Cæsarean section	0	0	5	1

To sum up: While a uterine scar greatly impairs the efficiency of a parturient, if the indication for the first Cæsarean section is not a recurring one, a trial of labour should be made in a hospital where a repeated Cæsarean section can be performed. This requires careful, constant and skilful supervision so that a full and thorough trial can be safely made.

A successful vaginal delivery on one occasion does not mean that all subsequent deliveries will be so.

Of course, when more than one Cæsarean section has been performed, it must be only in the exceptional case, as when premature labour occurs, that any labour should be allowed.

Previous Cæsarean Section and Disproportion.

There were 51 cases in which both previous Cæsarean section and disproportion were given as the first two indications, either being the first listed. In these cases the outlook for the infant was good.

No trial of labour was given. Two babies died, one (weighing five pounds) from atelectasis, and one (weighing six pounds 13 ounces) from a unilateral pneumothorax. Though the numbers are small, this perinatal mortality rate of 4% accentuates that given by King *et alii* (1953)—namely, 1% in 1003 Cæsarean sections in which the fœtal prognosis was excellent.

Placenta Prævia.

Placenta prævia was the first listed indication in 19% of Cæsarean sections in the Crown Street series, 1949 to 1954.

In the period from 1937 to 1945, Macafee (1945) had charge of all the patients with *placenta prævia* in the wards of the Royal Maternity Hospital, Belfast. The report of his meticulous study of these and the results of his scientific and skilful care of the patients are now history.

The results from the Women's Hospital, Crown Street, where the patients are distributed amongst all members of the staff, compare unfavourably with Macafee's results of over ten years ago. Two five-year periods at the hospital have been taken for comparison—the first up to a time ten months after Macafee's first paper was published, and the other commencing almost four years after its publication. Table XIV shows the figures at this hospital for

booked and unbooked patients with *placenta prævia* delivered by any method, and compares them with Macafee's series.

Browne (1955) has stated that "Macafee's low fœtal mortality was due chiefly, if not entirely, to his high Cæsarean Section rate". With no increase in the Cæsarean section rate at Crown Street, the perinatal mortality rate has been reduced to near that of Macafee.

Other factors must have been at work, such as the following: (i) better condition of patients reaching hospital; (ii) increased use of blood transfusion; (iii) the practice of Macafee's teaching to institute conservative treatment at first to allow the infants to become more mature; (iv) as taught by Macafee, better technique in Cæsarean section for *placenta prævia*; (v) better pædiatric care of infants.

TABLE XIV.

Hospital.	Years.	Number of Patients with *Placenta Prævia.*	Perinatal Mortality Rate.	Cæsarean Section Rate.
Royal Maternity Hospital, Belfast	1937–1945	191	23·5%	40·0%
The Women's Hospital, Crown Street ..	1941–1946 1949–1954	138 202	58·0% 26·8%	29·0% 29·0%

In Macafee's series (1949) the perinatal death rate following Cæsarean section was 7·1%.

Of the 59 babies delivered by Cæsarean section in cases of *placenta prævia* at Crown Street, 15 (25·4%) suffered perinatal death. The causes of these 15 perinatal deaths are shown in Table XV.

TABLE XV.

Post-Mortem Findings of Causes of Perinatal Death after Cæsarean Section or Placenta Prævia, 1949 to 1954.

Cause of Death.	Number of Infants.	Weight of Infants. (Pounds and Ounces.)
Emphysema	2	8, 0; 8, 0.
Atelectasis	5	3, 0; 3, 0; 3, 3; 3, 7; 4, 9; 4, 12.
Vernix pneumonia	1	3, 7.
Cerebral œdema	1	3, 14.
Suppurative meningitis ..	1	5, 12.
Spina bifida	1	5, 5.
Congenital atresia of trachea	1	6, 12.
Anæmia	2	2, 10; 4, 5.
Probably anæmia[1]	1	4, 10.

[1] No post-mortem was carried out; the mother had received 17 bottles of blood by transfus on before delivery.

Two infants were malformed, and 10 were premature, six of these weighing under four pounds.

That this high perinatal death rate is not unique is shown by the reports from the Women's Hospital, Melbourne, for the two years 1952 to 1954. In this period 127 patients with *placenta prævia* were treated, with a perinatal death rate of 22·8%. Cæsarean section was performed on 70 (55·1%), with 15 (21·4%) perinatal deaths. Prematurity is associated with many of these perinatal deaths.

In case it is still thought that to increase the Cæsarean section rate alone will decrease the perinatal mortality rate in *placenta prævia*, let us return to Macafee, who makes the following statement:

> It is very easy, but quite unjustifiable to perform a Cæsarean Section on every case of Placenta Prævia without considering all the factors in each case.

Elderly Primiparæ.

The policy at the hospital has been not to perform a Cæsarean section on elderly *primiparæ* unless there has been a trial of labour or unless there is some other indication.

During the years from 1949 to 1954, 18 Cæsarean sections (5·5%) were performed with the prime indication "elderly *primipara*"; the average age of these patients was forty-two years.

TABLE XVI.

Prematurity Associated with Perinatal Deaths Following Cæsarean Section for Placenta Prævia.

Hospital.	Number of Perinatal Deaths.	Number of Premature Infants.
Women's Hospital, Melbourne, 1952–1954 ..	15	8
Women's Hospital, Crown Street, 1949–1954	15	12

The second indication in each case is listed in Table XVII.

Induction of Labour.

No matter what the indication, labour should not be induced surgically unless one is prepared to consider performing a Cæsarean section if labour cannot be initiated within seventy-two hours. To allow things to drift longer than this greatly prejudices the chances of the infant.

TABLE XVII.

Elderly Primiparæ: List of Second Indications for Cæsarean Section.

Second Indication.	Number of Cases.
Hypertensive toxæmia	3
Hypertensive toxæmia, unsuccessful induc- of labour (mechanical rupture of membrance) three days earlier	1
Fœtal distress	1
Fœtal distress, 15 years' sterility ..	1
Sterility	2
Breech presentation, 13 years' sterility	1
Breech presentation, Parkinson's disease	1
Breech presentation, contracted pelvis ..	1
Failure of head to engage, owing to fibroid tumour	1
Unsuccessful trial of labour (12 hours, 42 hours)	2
Flat, android pelvis (five hours' trial of labour)	1
Slight disproportion	1
Post-maturity, disproportion (baby weighed nine pounds eight ounces) ..	1
Previous rupture of bladder	1
Total	18

In this connexion it is of interest to note that Browne (1952) has shown that there is no serious delay in the onset of labour with an "unripe" cervix compared with the onset with a "ripe" one, and holds that the "unripe" cervix does not contraindicate the performance of a surgical induction of labour.

Contraindications to Cæsarean Section.

I know of no absolute contraindication to Cæsarean section. There are several partial ones.

Dead Fœtus.

In the five years from 1949 to 1954 at the Women's Hospital, Crown Street, no Cæsarean section has been performed when the fœtus was known to be dead. Peel (1955) has given some rare instances in obstructed labour when Cæsarean section may be indicated.

Gross Sepsis of Abdominal Wall or Genital Tract.

Every endeavour should be made to avoid Cæsarean section in the presence of gross sepsis of the abdominal wall or genital tract.

Prematurity of the Fœtus.

Prematurity of the fœtus as a contraindication applies only when an elective Cæsarean section is contemplated. Here it is of the utmost importance to be certain that the fœtus is mature. An X-ray examination for maturity is an additional safeguard.

Prematurity is the most important associated cause of perinatal death following Cæsarean section.

At Crown Street, in the period 1949 to 1954, there were 28 perinatal deaths associated with Cæsarean section. Half of these infants (14) weighed under five pounds, and three-quarters (21) were premature.

In no case was the premature birth due to an error by the obstetrician in gauging the maturity of the infant. The reasons why that particular time was chosen for the operation are set out in Table XVIII.

TABLE XVIII.

Indication for Cæsarean Section.	Number of Perinatal Deaths.
Ante-partum hæmorrhage	15
Preeclampsia	3
Previous Cæsarean section—onset of labour	1
Previous Cæsarean section—tender scar	1
Elderly *primipara*—rupture of membranes twelve hours previously	1
Total	21

Gross Malformation of the Fœtus.

To try to avoid delivering a malformed fœtus by Cæsarean section, all patients should undergo an X-ray examination for fœtal abnormality prior to the operation. This particularly applies to patients with *placenta prævia*, in whom the malformation rate is increased. In cases of disproportion one must always exclude hydrocephalus as a cause of the disproportion. In the year 1953-1954, 46 Cæsarean sections were performed at Crown Street. In at least 16 of these cases an X-ray examination was made within a few weeks of delivery for one of the following reasons: multiple pregnancy, position, pelvimetry, placentography, maturity or normality of the fœtus. It was unusual for a specific request for a report on normality of the fœtus to be made, and rare for one to be given unless it was requested. The radiologist may have difficulty in detecting some of the bony fœtal abnormalities in the fœtus *in utero*, such as a mild degree of hydrocephalus, or as in one case, bony defects in the cranial bones; but if he is expressly asked for an opinion on normality of the fœtus, he is less likely to miss anything.

Summary.

1. There is an appreciable maternal mortality after Cæsarean section.

2. The infantile perinatal mortality rate is in the region of 4·3%, with the exclusion of cases in which *placenta prævia* was the indication, and 8·5% when all cases are included.

3. A decline in fecundity follows Cæsarean section.

4. Mechanical dystocia, uterine scar or hæmorrhage is the first listed indication for over 70% of Cæsarean sections performed at the Women's Hospital, Crown Street.

5. A decrease in the incidence of eclampsia led to a decrease in the number of Cæsarean sections performed for preeclamptic toxæmia, thus almost eliminating one of the major indications for Cæsarean section.

6. In four years only four Cæsarean sections have been performed for preeclamptic toxæmia.

7. Patients with a true conjugate of more than 3·5 inches (8·4 centimetres) should be given a trial of labour.

8. In four years 134 trials of labour were made. In those lasting over twenty-four hours the Cæsarean section rate was 26·5%. The perinatal mortality rate for the whole series was 4·5% (including three cases of malformation of the fœtus).

9. From the fœtal point of view, prolonged labour (over forty-eight hours) is to be feared; the perinatal mortality rate is 10%.

10. The Cæsarean section rate at the Women's Hospital, Crown Street, in the years from 1949 to 1954 has fallen from 1·9% to 1·0%. One-third of the patients had previously undergone Cæsarean section.

11. In patients who had undergone one previous Cæsarean section, over two-thirds were delivered vaginally.

12. Over the period only one scar ruptured (0·5%). This patient was not being given a trial of labour; the scar of a high Cæsarean section ruptured at the onset of labour.

13. In the period 1949 to 1954, the perinatal mortality rate following *placenta prævia* was 26·8%, compared with 58% for the years 1941 to 1946, the Cæsarean section rate in this condition remaining unaltered at 29%. Of the perinatal deaths, 80% were associated with prematurity.

14. "Elderly *primipara*" by itself is not an indication for Cæsarean section.

15. No matter what the indication, labour should not be induced surgically unless one is prepared to consider performing a Cæsarean section if labour cannot be initiated within seventy-two hours.

16. The partial contraindications to Cæsarean section are death of the fœtus, gross sepsis, gross malformation of the fœtus and prematurity.

17. In the series of 327 Cæsarean sections there were 28 perinatal deaths. Three-quarters of these were associated with prematurity due to a severe maternal condition, and none was due to an error in estimating maturity on the part of the obstetrician.

18. Before Cæsarean section is performed, all patients should be radiologically examined to try to exclude fœtal abnormality.

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CHLORPROMAZINE AND RESERPINE IN THE TREATMENT OF CHRONIC MENTAL ILLNESS.

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A PRELIMINARY report on the subject of chlorpromazine and reserpine in the treatment of chronic mental illness appeared in this journal in July, 1955 (Morgan, 1955). With a year's experience further evaluation of this treatment can now be given.

The results obtained in only six of the chronic wards of this hospital will be described, for two major reasons. The first is that the patients selected had all been regarded as hopelessly insane and had been in the Mental Hospital, Parramatta, for at least a year, several having been in a mental hospital for over thirty years. Secondly, such good results have been achieved in the treatment of patients newly admitted to the hospital that the percentage of "recoveries" would have tended to make the results seem even better than they are so far as chronic cases are concerned.

The object of this paper is to emphasize just what can be done for patients who were regarded as having a hopeless prognosis, and for whom all previous treatments have been of no avail; and it should be stressed that each patient's progress has been assessed with extreme conservatism.

A great wealth of research has been carried out, both in Australia and overseas, on the subject of the treatment of psychiatric patients with chlorpromazine and reserpine, and it is now possible to state that findings here closely parallel those recorded by, for example, Kinross-Wright (1955*a*) in the United States of America.

By far the most comprehensive publication on the use of chlorpromazine, and more particularly of reserpine, is in the *Annals of the New York Academy of Sciences* of April 15, 1955, and those interested will be well rewarded by a study of the many articles and references to the electropharmacological effects of these drugs and the clinical experiences recorded by the different investigators.

The Drugs.

Most psychiatrists in Australia are now familiar with the origins of chlorpromazine and reserpine and with their formulæ (Ramsay Webb, 1955; Bower, 1955), but a brief recapitulation may be helpful.

The synthesis of chlorpromazine ("Largactil", "Thorazine" or "Megaphen") was the outcome of nearly twenty years' work by French investigators, who were engaged in research into what they termed the "syndrome of irritation" or "the phenomena of Reilly". It was shown by these workers that chlorpromazine by itself was able to counteract the effects of sympathetic irritation and modify the pituitary-adrenal mechanism's response to aggression, and it was postulated that chlorpromazine exerted its influence through the brain-stem reticular formation (Hopkin, 1955).

Chlorpromazine is a phenothiazine derivative closely related to ethopropazine hydrochloride ("Lysivane") and the antihistaminic promethazine hydrochloride ("Phenergan").

There is now intense activity overseas to discover other phenothiazine derivatives which can modify mental behaviour, and one example is N-methylpiperidyl-3-methylphenothiazine ("Pacatal"), which is being investigated in this hospital at the present time.

The romantic background to the discovery of reserpine is highlighted by the fact that in 1665 Dr. Nathan Hodges mentioned the use of "snake root" in treating the plague—but this use of the drug does not seem to have been very successful. However, it was not until the autumn of 1953 that Swiss workers isolated reserpine (which was called "Serpasil" by Ciba) from the Indian snake root or, more correctly, *Rauwolfia serpentina*.

Altogether, 14 different pharmacologically active alkaloids have been isolated from *Rauwolfia serpentina*, including the hallucinogen yohimbine. This has a vaguely similar formula to that of reserpine.

It has been shown that reserpine is the alkaloid primarily responsible for the action of the whole root of *Rauwolfia serpentina*, whilst another alkaloid, rescinnamine, has a similar but weaker action.

More recently "Deserpidine" (desmethoxyreserpine), obtained from *Rauwolfia canescens*, has been shown to have a pharmacological action which lies within the same range as that of reserpine; but apart from this it seems that no useful purpose can be achieved by using partial fractionations of rauwolfia for treating mental illness when the active ingredient—chemically pure and uniformly standardized—is readily available in reserpine.

The site of action of reserpine, as of chlorpromazine, is thought to be in the mid-brain reticular formation, for it increases cortical inhibition of diencephalic structures. The probable explanation of this action, in the case of reserpine, is that the drug facilitates synaptic transmission.

Indications.

As a result of experience, earlier impressions as to what are the appropriate indications for the exhibition of chlorpromazine and reserpine have been confirmed.

In the main, chlorpromazine is used for patients who show unpredictable motor restlessness, violent homicidal behaviour and violent aggression, whilst some success has been achieved with chlorpromazine in the treatment of depressives. In general, however, it has proved disappointing in this last-mentioned field. Nevertheless, it might be germane to mention that Dr. John Lisyak, in the male admission ward of this hospital, has had a number of encouraging successes with chlorpromazine in the treatment of depressive states.

However, before successes are claimed for any form of treatment of cyclic insanities, one should not overlook the fact that, from the very nature of the disease, there is a possibility of natural remissions, and these may render difficult the true assessment of the value of the treatment concerned.

Reserpine is used for patients whose thought processes and associations are inappropriate, who are tormented by their hallucinations, and who show regressive behaviour. It has been found to be of particular benefit in cases of paranoid schizophrenia and paraphrenia—illnesses which, in the past, have been notoriously difficult to treat successfully. The same degree of success has not been obtained with reserpine in simple and hebephrenic schizophrenia, although several hebephrenics, previously treated in a voluntary clinic in Sydney without avail and then sent to this hospital under certificates, have achieved an astonishing clinical remission.

One of the most remarkable mental effects of both reserpine and chlorpromazine is the manner in which patients may without psychotherapy suddenly lose their paranoid ideas and gain insight into their mental state. Frequently patients will say how silly their previous thoughts were, and how they cannot understand how they came to believe that people were against them or talking about them; and these patients spontaneously comment that they really must have been ill without realizing it.

It will be seen from the tables incorporated in this article that 30 patients were treated with a combination of both chlorpromazine and reserpine. In the first place these patients were started on the drug considered most likely to benefit them; and if at the end of two months no clinical improvement resulted, then the other drug was tried. If this too failed, then a combination of the two drugs was given, and this combined therapy has had a limited success. To illustrate this point the case of one patient is interesting. This patient had not responded to massive doses of either drug separately, but after five weeks of combined therapy suddenly showed considerable improvement, which has since been maintained.

Thus it will be seen that adequate treatment with these drugs, although at times rapidly dramatic, is in general a far slower process than the quasi-magical pushing of the button in electroconvulsive therapy. On the other hand, the drugs have brought about a clinical remission in patients who had failed to respond to all other forms of treatment, and for whom without these drugs there would have been no other alternative but a leucotomy, with its somewhat unpredictable results.

It will be noticed that the term "clinical remission" has been used rather than "cure"; for it will be many years before a cure can be claimed, if ever. Nevertheless these drugs have rendered these patients symptom-free—and some have been so for many months. It is believed that ultimate cure may depend on whether these patients become accustomed to feeling well and establish new habit patterns of rational behaviour. This will take time, and some patients may never achieve this change. It seems likely, too, that some patients may always have to rely on these drugs to maintain their remission.

By far the most difficult thing to establish in some of these schizophrenics who have achieved a pronounced improvement is the ability to form sound, rational judgements about the various complexities of everyday living, complexities which are taken for granted by those who enjoy normal mental health. One wonders, indeed, if it will ever be possible to create common sense in an adult if this has not been acquired during the process of maturation.

Clinical Material and Methods.

This report deals with the results obtained in the treatment of 250 female patients suffering from chronic mental illness.

When it was first decided to use chlorpromazine and reserpine on the chronically affected patients in this hospital, those patients showing the worst behaviour problems were selected by the medical superintendent, Dr. Eric T. Hilliard, who, because of his many years' experience in the hospital, knew the behaviour-patterns of these patients intimately. It was because these apparently demented patients in our chronic wards did improve so miraculously that the preliminary report was published, in the hope that it would stimulate and encourage others to give a clinical trial to these compounds on their own patients, without waiting until a statistical evaluation covering the patients treated in this hospital proved practicable.

Different dosages of the two drugs have been used at different times, and experience has modified the dosages described in the previous article. The largest dosage of chlorpromazine used on any patient has been 1800 milligrammes per day by mouth; and, strange to say, except for a half-closing of the eyelids, this patient remained surprisingly alert, although in the main devoid of spontaneous movements.

The largest dose of reserpine has been 30 milligrammes a day on one patient; but she developed a mask-like facies with excess salivation, a bowed stance, and arms held by the side slightly flexed but without tremor. As the patient was unable to care for herself properly, the dosage was reduced.

The dosages now adopted have been arrived at after due deliberation and after consideration of several factors. The first aim is to achieve the maximum clinical response as speedily as possible. Secondly, the experience of competent overseas psychiatrists has confirmed these dosages. Thirdly, it was necessary to adopt a standard dosage which could be readily followed by the nursing staff and which would be standard throughout the wards.

Reserpine is now issued to the wards only in one-milligramme tablets and in one-milligramme ampoules (Ciba), the intramuscular or intravenous route being reserved for patients who are non-cooperative and who refuse to take tablets. Initially the intramuscular route is the most efficacious; but with a large number of patients this method of administration was beyond the capacity of the staff available.

On the first day the patient receives four milligrammes of reserpine (that is, two tablets each of one milligramme twice a day); on the second day eight milligrammes (four tablets twice a day); and on the third day 12 milligrammes (six tablets twice a day). This third-day dosage is maintained until a clinical response is obtained; by this is implied not merely drowsiness and slowness of movement, but also evidence that the patient is more realistically in contact with the environment and that the symptoms are lessening. When this stage is reached, or in any case at the end of three weeks, the dosage is reduced by not more than four milligrammes a day to a maintenance dosage which, in the vast majority of cases, has been found to lie between two and four milligrammes per day.

Chlorpromazine is administered according to a similar schedule; but in this case "100 milligrammes" should be substituted for the "one milligramme" given above. This is done for the sake of uniformity and to avoid confusion should nurses be transferred from one ward to another.

Chlorpromazine ("Largactil") is supplied in 100-milligramme tablets and 50-milligramme ampoules (May and Baker), and so on the first day the patient receives an oral dose of 400 milligrammes, on the second day 800 milligrammes, and on the third day 1200 milligrammes (that is, six tablets twice a day) until a clinical response is obtained, when the dosage is reduced in a similar manner to that outlined above, to a maintenance dose, which is generally of the nature of 200 to 400 milligrammes per day.

Of course, with both reserpine and chlorpromazine it may be that on the second day the clinical improvement is such that there are no grounds for further increasing the dose beyond four tablets morning and evening. Each patient's progress is therefore discussed with the sister in charge of the ward, and future treatment is determined in the light of this clinical evaluation.

This scheme of administration may differ from that employed by others; but in this hospital it has given results which are most encouraging. It is, of course, possible that further experience may cause a modification of this standard procedure; but at the moment this does not appear likely.

When chlorpromazine and reserpine are used concurrently it is usual to employ half the standard dosage of each. Thus on the third day such a patient would receive six milligrammes of reserpine and 600 milligrammes of chlorpromazine—still a total of 12 tablets—that is, three tablets of each drug morning and evening. There seems to be no advantage in using larger doses than those outlined above, since beyond this range there is a greater possibility of producing a syndrome which resembles Parkinsonism; this can be disturbing to the patient, even when partially controlled with ethopropazine hydrochloride ("Lysivane"). Some authors have suggested that it is necessary to produce a clinical response of this nature in order to achieve a maximum therapeutic result; but this has not been found necessary in the present series.

Results.

The behaviour of each patient was recorded on an individual standard "behaviour and treatment" form used for many years in this department. This lists behaviour under the following headings:

Solitary	Destructive	Mute	Delusional
Suspicious	Confused	Talkative	Hallucinated
Resistive	Dull	Incoherent	Faulty
Impulsive	Agitated	Noisy	Perverted Habits
Aggressive	Restless	Depressed.	Idle

Behaviour was recorded by the figures 4, 3, 2, 1, 0 against each heading, 4 representing the maximum degree of disturbance and 0 the absence or complete disappearance of the symptom concerned. At the end of the year all these individual charts were analysed, in collaboration with the sisters in charge of the wards, and the findings were utilized in the preparation of the tables included in this article.

The assessment of the degrees of improvement has been on a very conservative basis, and in the main has been that of the nursing staff, who are in close contact with the patients. It can be stated that no single patient was made worse by treatment, and every patient classified as "unchanged" has made at least some slight improvement, although not sufficient to qualify for the designation "improved".

Actually results have been classified under the following headings: (i) "Unchanged"; this has been described earlier. (ii) "Improved"; under this heading are included patients who are now quiet, clean and able to care for themselves. (iii) "Markedly improved"; this designation has been applied to patients who are now able to attend all recreations and help in the wards, and are bright, cooperative and friendly; (iv) "clinical remission"; under this heading have been listed those patients who have proceeded on leave or who are mentally well enough to do so as soon as suitable arrangements can be made, and who are symptom-free.

Many patients classified as "markedly improved" have had days or week-ends away from the hospital or have even gone home to their families; but the patients still retain some symptoms of their former illness, and therefore have not attained a clinical remission.

Table I shows the length of residence of the 250 patients in a mental hospital, thirty-six years being the greatest length of residence, and no patient having been in hospital for less than one year.

TABLE I.
Length of Residence.

Years.	Number on Chlorpromazine Treatment.	Number on Reserpine Treatment.	Number on Chlorpromazine and Reserpine Treatment.	Totals.
1 to 2 ..	22	11	6	39
2 to 3 ..	20	18	3	41
4 to 5 ..	24	10	5	39
6 to 10 ..	25	7	8	40
11 to 15 ..	20	6	1	27
16 to 20 ..	20	10	2	32
21 to 25 ..	12	2	5	19
26 to 30 ..	6	1	0	7
Over 30	4	2	0	6
Total	153	67	30	250

Table II shows the age distribution of the patients, the youngest being aged nineteen years and the oldest eighty-three years.

Table III shows the changes produced by the drugs; and although only 19% of the patients achieved what could be regarded as a clinical remission, it will be seen that, if those who are "markedly improved" are included, 49% of the patients have shown a tremendous improvement over their former state.

It will be noted from Table III that of the 30 patients who had failed to respond to either drug separately, six were helped to a great degree by a combination of the two drugs.

Table IV shows the diagnoses in respect of the 250 patients, two-thirds of whom were either schizophrenics or paraphrenics. It was not found possible to break down the schizophrenics into paranoid, simple *et cetera*, because some had been admitted to hospital so many years earlier that they were classified as suffering from *dementia præcox* or acute delusional insanity, in accordance with the concepts of those days. Table IV also shows how many patients were treated with each drug or a combination of both. It will be noted that the treatment of depressives with reserpine was avoided, depression being considered a contraindication to the exhibition of this drug.

TABLE II.
Age Groups.

Age (Years).	Number on Chlorpromazine Treatment.	Number on Reserpine Treatment.	Number on Chlorpromazine and Reserpine Treatment.	Total.
Under 20 ..	2	0	1	3
Under 30 ..	14	5	4	23
Under 40 ..	27	12	8	47
Under 50 ..	46	22	7	75
Under 60 ..	34	16	7	57
Under 70 ..	21	9	1	31
Under 80 ..	8	3	2	13
Under 90 ..	1	0	0	1
Total	153	67	30	250

Of the three patients suffering from *dementia paralytica*, one showed no improvement, one was improved and one much improved; and of the nine mental defectives, six were improved, two were much improved and one has gone on leave.

One of the most interesting cases is that of the patient suffering from an obsessive-compulsive neurosis.

TABLE III.
Degree of Change for All 250 Patients.

Treatment.	Unchanged.	Improved.	Markedly Improved.	Clinical Remission.	Total.
Chlorpromazine	25	49	51	28	153 (61%)
Reserpine ..	11	19	19	18	67 (27%)
Chlorpromazine and reserpine	12	12	4	2	30 (12%)
Total ..	48 (19%)	80 (32%)	74 (30%)	48 (19%)	250

This woman, aged forty-six years, admitted to hospital in 1954, had for some years been impelled to wash her hands almost without cessation; and each time she used the toilet she would fill and choke it with paper, these symptoms being manifestations of guilt over a previous love affair. In spite of a good education, she was completely impervious to psychotherapy or, indeed, to any other form of therapy. She had, in fact, had courses of electroconvulsive and full coma insulin therapy without avail. Eventually she underwent a bilateral prefrontal leucotomy, without any improvement whatsoever.

On her admission to hospital, the patient was in a pitiable plight—thin, emaciated and restless; and her continual handwashing and successful blocking of the ward toilets were a tremendous problem. She was a wretched, unhappy woman for whom there seemed no hope. Her transformation following treatment with chlorpromazine was almost unbelievable. She became relaxed and happy and had a splendid appetite, gaining considerable weight, and she laughed at her former symptoms. After several months she proceeded on leave and obtained a position as a governess to some children.

It was surprising when this patient returned to the hospital, with all her old symptoms in full force. On inquiry, it was found that, after seven months, she had decided that she was so well that she no longer needed to take the tablets, and her relapse was the result of this decision. She has again been treated with chlorpromazine, and is once more ready to proceed on leave.

TABLE IV.
Diagnosis.

Diagnosis.	Number on Chlorpromazine Treatment.	Number on Reserpine Treatment.	Number on Chlorpromazine and Reserpine Treatment.	Total.
Schizophrenia ..	68	39	19	126
Paraphrenia ..	23	23	4	50
Affective psychoses:				
(*a*) Manic ..	8	1	1	10
(*b*) Mixed ..	1	1	1	3
(*c*) Depressive ..	12	0	3	15
Congenital mental deficiency ..	9	0	0	9
Congenital mental deficiency with epilepsy ..	9	0	0	9
Epileptic psychosis	9	0	0	9
Confusional psychosis	6	0	1	7
Dementia (atherosclerotic) ..	4	3	1	8
Dementia paralytica	3	0	0	3
Obsessive-compulsive neurosis ..	1	0	0	1
Total	153	67	30	250

Of the 18 patients who suffered from epilepsy and who were treated with chlorpromazine, the condition of seven was "unchanged", that of six "improved", and that of five "markedly improved". Although only two of them improved to the extent of going home on leave, the majority have become much more pleasant in the wards. No patient receiving the drug showed an increase in the number of convulsive seizures per month, which in any case was very small.

TABLE V.
Change in 126 Schizophrenics.

Treatment.	Unchanged.	Improved.	Markedly Improved.	Clinical Remission.	Total.
Chlorpromazine	13	21	21	13	68 (54%)
Reserpine ..	3	15	11	10	39 (31%)
Chlorpromazine and reserpine	9	8	1	1	19 (15%)
Total ..	25 (20%)	44 (35%)	33 (26%)	24 (19%)	126

Table V shows the change in the 126 schizophrenics, and Table VI the change in the 50 paraphrenics in the series.

It must be remembered that in general the schizophrenics had deteriorated further than the paraphrenics and might have been classified as suffering from "secondary dementia"—a diagnosis which should be strenuously avoided, for, as Noyes (1948) suggests, this end-result of the schizophrenic process is a "habit deterioration" rather than "mental deterioration", the term frequently employed. For example, one of our patients who has been apparently utterly deteriorated and demented for twenty-two years is one of those who has shown the greatest clinical improvement.

Table VII shows the change in 28 patients with affective psychoses. The information in this table is not divided into component manic, mixed and depressive phases, because in cyclothymia the patient may change from one phase to another, and so the diagnosis in the records depends on the state of the patient on admission to hospital and not at the present time. However, all these patients were considered to present behaviour problems.

It will be seen from Table VII that the patients with affective psychoses were improved far more by chlorpromazine than by reserpine, as had been suspected earlier; but, as has already been mentioned, the use of reserpine was avoided in the treatment of patients with depressive states.

TABLE VI.
Change in 50 Paraphrenics.

Treatment.	Unchanged.	Improved.	Markedly Improved.	Clinical Remission.	Total.
Chlorpromazine	1	6	14	2	23 (46%)
Reserpine ..	6	4	7	6	23 (46%)
Chlorpromazine and reserpine	0	2	1	1	4 (8%)
Total ..	7 (14%)	12 (24%)	22 (44%)	9 (18%)	50

Table VIII shows the length of time during which patients were in hospital prior to being able to go on leave.

Table VIII shows that 41 patients have already gone home to their families, out of the total 250 patients treated; and nine more will do so as soon as suitable arrangements can be made. Under the *Lunacy Act* in New South Wales it is customary for each patient who leaves hospital to be on long leave for a period of twelve months, and when a medical practitioner certifies that he is well, he is discharged completely from the hospital. Because of this no long-term results of treatment can yet be given.

TABLE VII.
Change in 28 Patients with Affective Psychoses.

Treatment.	Unchanged.	Improved.	Markedly Improved.	Clinical Remission.	Total.
Chlorpromazine	2	4	5	10	21 (75%)
Reserpine ..	1	1	0	0	2 (7%)
Chlorpromazine and reserpine	1	2	2	0	5 (18%)
Total ..	4 (14%)	7 (25%)	7 (25%)	10 (36%)	28

The state of the wards before this treatment was instituted has been described previously (Morgan, 1955); and even now it is a little strange to pass through formerly turbulent wards and find them quiet and peaceful, with patients sitting about reading, sewing or playing cards. There have been a number of visitors to the wards who knew them previously, and one and all have been most impressed with the change.

Prior to the availability of chlorpromazine and reserpine, when a patient was newly admitted to the hospital in a disturbed and restless state, it was occasionally suggested that she be sent to a so-called "refractory ward". This is no longer a possibility, as there are now no such wards. The need for them has passed.

Since the advent of chlorpromazine and reserpine, sedatives are seldom required and paraldehyde is rarely used, although previously 30 to 40 draughts of paraldehyde were

being administered each evening. Now, should a patient require sedation, it is customary to use only "Sodium Amytal" or "Nembutal".

There has also been a great reduction in the use of electroconvulsive therapy. Formerly 35 to 40 patients were given electroconvulsive therapy twice a week, but since July, 1955, this method of treatment has been completely discarded in these chronic cases. As a matter of interest, this method of treatment now has a very minor role even in the admission wards. In fact, no electroconvulsive therapy has been given in the male admission ward of the hospital since November, 1955, and at the present time there are only two patients out of 65 in the female admission ward who are receiving this treatment.

TABLE VIII.

Length of Residence in Hospital of Patients Proceeding on Leave.

Stay in Hospital.	On Long Leave.	Able to go on Long Leave, but Still in Hospital.	Daily or Week-end Leave.
Under 3 years	18	3	5
Over 3 and under 5 years	10	2	8
Over 5 and under 10 years	6	3	4
Over 10 and under 15 years	6	0	3
Over 15 and under 20 years	0	0	2
Over 20 years	1	1	2
Total	41	9	24

Side Effects.

In general, it may be said that patients who receive reserpine do not develop so many side effects as those who receive chlorpromazine. The side effects have been dealt with in a paper published earlier this year (Morgan, 1956*b*).

Discussion.

Experiences in this hospital with chlorpromazine and reserpine parallel those of Goldman (1955) and of Kinross-Wright (1955*c*) with chlorpromazine, and of Kinross-Wright (1955*a*) with chlorpromazine and reserpine; yet others, in England and in this country, do not seem to have had the same encouraging results. There may be several reasons for this; perhaps the most important single factor is hesitation on the part of some workers to use sufficiently large doses of the drugs. For example, the results achieved in this hospital have been very much better with the larger doses now used than they were with the smaller doses used originally.

It is essential to have the whole-hearted cooperation of the nursing staff, for on them depends the ultimate responsibility of seeing that each patient receives the right quantity of the drug prescribed, especially as some patients become adept at appearing to take tablets when, in actual face, the tablets are hidden under the tongue.

Before any new treatment is implemented in this hospital the object of the treatment is explained to the nursing staff and they are told what may be expected of it and what side effects to watch for. This gives them a sense of participation, without which their cooperation would be hard to gain, and in this hospital we have been fortunate in receiving the whole-hearted support of our staff.

Another point is that over many years the Mental Hospital, Parramatta, has been regarded as a "maximum security" hospital, and difficult and violent patients have been sent here from country hospitals; whereas in some hospitals the majority of patients are quiet, apathetic and self-absorbed, and these last-mentioned patients do not respond as well to treatment as do patients who exhibit tension, aggression and an excess motor activity.

That these drugs have brought a real advance in the treatment of chronic mental illness is undoubted, and 80% of our patients have shown improvement following their use.

It is true that the cost of using large quantities of these drugs is considerable; but there have been savings in other directions. Even if only one patient had been returned to the community the expenditure of the money would be justified from the purely humanitarian point of view, especially when it is considered that too little has been spent on the mentally ill in the past.

With regard to the economic point of view, it is of interest that the hospital tailor has not made a canvas rug or jacket since June, 1955, that considerably less crockery is being broken, and that very little linen and clothing is now destroyed, beyond ordinary wear and tear, while far less soiled linen is sent to the laundry. These are all positive savings.

When it is remembered that, according to an American report, the average length of stay of a schizophrenic in a mental hospital is thirteen years, and that in 1954-1955 it cost £284 per year to keep a patient in this hospital, the saving to the taxpayer from the rehabilitation of the 23 schizophrenics only, who have achieved a clinical remission, is approximately £6432 *per annum*.

Dr. E. Cunningham Dax, Chairman of the Victorian Mental Hygiene Authority, put it another way when he recently stated in Sydney that the cost to the community for each patient who spends a lifetime in a mental hospital, including the loss of earning capacity *et cetera*, is about £30,000; on this basis the early rehabilitation of 23 schizophrenics might be estimated to have saved the community nearly £700,000.

Another gratifying result of this treatment is that there are now no injuries either to fellow patients or to nurses, with a consequent easing of the burden on the staff generally. Furthermore, there is a new feeling of pride in the nursing staff—they can now see really encouraging results for their labours, and they rejoice in their new role of therapists. In this connexion the comments of Noce, Williams and Rapaport (1955) are relevant. These authors stress the need for a "total push" programme in conjunction with drug treatment, in which the efforts of psychiatrists, psychologists, social workers, rehabilitation therapists and nurses are combined in a team approach. Although this has not yet been possible in this hospital, it is believed that such an approach is essential for the maximum benefit from the treatment to be reaped by each patient. While this approach would initially cost the taxpayer more, it would in the end result in a great saving of public moneys.

Chlorpromazine and reserpine have proved superior to electroconvulsive therapy, but are slower in bringing about a remission of symptoms. However, when this remission is achieved the patients appear to have a better insight into their illness and, above all, there is not the unreasoning fear that many have of electroconvulsive therapy, when instead they are asked only to take a few tablets.

It has been shown that many side effects occur with these drugs; and yet, although some have caused concern, there seems to be little risk to life. This is of importance, especially when the effect of these drugs is contrasted with that of full coma insulin therapy or leucotomy.

In the present state of our knowledge, with new drugs being discovered and as yet relatively untried, it is held that it would be amoral to consider subjecting a patient to leucotomy before all other forms of treatment had been tried exhaustively and found to be unsuccessful.

From the experience gained in treating this series of patients it seems that these drugs exert a beneficial effect on a wider range of mental illnesses than any other form of therapy so far available, and whilst as yet there is no uniformity of opinion on what are the appropriate indications for the use of each drug, it is likely that this, too, will soon be placed on a rational basis.

There are at present some very attractive theories advanced by Fabing (1955*b*) that abnormal breakdown of naturally occurring indoles in the body may ultimately be shown to be the cause of schizophrenic illness. This concept was mentioned in an earlier article (Morgan, 1955).

The question as to whether schizophrenia is metabolic in origin, chemically induced or of psychogenic origin as yet remains unanswered. However, these results show that many symptoms previously considered unchangeable are, after all, modifiable; and neuropharmacological research, such as is being conducted at the University of Sydney by Professor Peter Bishop and at Canberra by Professor John Eccles, may ultimately disclose a rational basis for "functional" mental illness.

Incidentally, Fabing (1955*a*) has also suggested the generic word "ataraxics" for drugs such as chlorpromazine, reserpine and azacyclonol (of which "Frenquel" is the hydrochloride), which bring about a "freedom from confusion". This seems a convenient term to describe what is, in many respects, a new concept in psychiatry.

Lastly, a word of caution should be given that, as with any new treatment, undue over-enthusiasm must be avoided at all costs, lest the undoubted value of the treatment be discredited by extravagant claims. It is believed that the opinions expressed in this article, based as they are on the actual results achieved, are a sober description of the changes that have occurred in the condition of the patients.

Summary.

The object of this paper is to show how chronic mental illness can be successfully modified in many cases by the use of chlorpromazine and reserpine. This improvement is not due simply to more interest being taken in the patients, but is due to the drugs themselves. The chemical nature of the drugs and their backgrounds are briefly outlined, and the appropriate indications for the optimum use of each drug are given. The methods adopted in using these drugs have been outlined in some detail; and the results have been given with tables to illustrate the degree of improvement achieved in 250 female patients suffering from chronic mental illness.

Occasional references have been made to the use of these drugs in the treatment of recently admitted patients, although these really do not come within the scope of this paper. However, this has been done only in order to clarify some particular point.

It has been shown that approximately 20% of the chronically insane patients have improved to the stage at which they can be considered fit to return to community life with a minimum of supervision.

Brief mention has been made of the economic and other aspects of the improve dstatus of the patients, and the side effects which may be encountered during the use of these drugs. Finally, some of the factors which may have influenced these results have been discussed, and the hope is expressed that in the future it may be possible to explain schizophrenia on a rational scientific basis.

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TUBERCULOSIS IN MELANESIAN NATIVES: A SUMMARY OF AUTOPSY FINDINGS FROM THE PRE-WAR ERA (1922 TO 1940).

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AT this stage in the antituberculosis activities that have followed the end of the war in the Pacific, it hardly seems necessary to detail the state of affairs that existed prior to that war. The known facts about tuberculosis in the native population are contained in the official reports of the period (1922-1940) and the few published papers based on them (Heydon, 1924, 1937, 1938; Clements, 1936). During the first decade after the war an awareness of the situation in the Pacific has led to extensive programmes of radiological examination, skin testing and B.C.G. immunization (North and Jamieson, 1950). These measures have been put into operation in some areas, and in general it may be said that the policy is one that aims at falling into line, as regards early diagnosis, treatment and prevention, with other antituberculosis campaigns in Australia and elsewhere.

In presenting this summary of pre-war autopsy findings at so late a date, the writer hopes that, in spite of their antiquity and inadequacy, they may be of some value in assessing possible changes in the pattern of the disease which may accompany an increasing level of immunity.

Heydon (1924), of whose work these records are a continuation, pointed out that although resistance to tuberculous processes was low in these people, it was not

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TABLE I.[1]

Subjects with Quiescent or Healed Tuberculous Lesions.

Reference Number.	Age in Years.	Cause of Death.	Brief Description of the Lesion.
		Subjects with Early Lesions in the Lungs.	
A/28	—	Dysentery.	Several caseous nodules in left lung.
A/190	28	Pneumonia.	Right lung: area of small grey tubercles, some confluent foci surrounded by fibrous tissue. Left lung: more advanced lesions, one small cavity with caseous matter, some scattered grey tubercles.
A/426	22	Dysentery.	Few small cavities both lungs, no miliary tubercles.
A/152	25	Bronchopneumonia.	Right lung: one caseous nodule. Bronchial glands caseous, Ziehl-Neelsen smear positive result.
31/25	33	Dysentery.	Area in one lung one inch by two inches thickly sown with minute tubercles surrounded by zone of fibrosis.
32/30	25	Suicide.	Both lungs: several caseating nodules in upper lobes with outlying grey tubercles.
34/48	25	Dysentery.	Apex of left lung: small nodular focus with some surrounding fibrosis. No dissemination in lung. Pretracheal and bronchial glands show tuberculous foci. Small infiltrated gland near root of aorta.
		Subjects with Quiescent or Obsolete Lesion in Lungs.	
H/148	33	Tropical ulcer.	Apex of right lung: old apparently quite healed tuberculous lesion consisting of cicatrized nodules and a few pin-head sized, dry, yellow foci.
H/211	27	Pneumococcal pericarditis, empyema.	Lungs: few small obsolete foci. Bronchial glands: few rather dry caseous foci both sides. No other tuberculous lesion in body.
30/66	30	Pneumonia.	Left apex: old puckered scar and some calcareous granules.
31/10	60	Tropical ulcer, gangrene.	Left lung: old fibrosed scar apex. No surrounding tubercles or caseation.
31/62	27	Pneumonia.	Right lung: old calcareous focus at apex.
32/91	50	——	Lungs: few sago-grain bodies. Caseous gland at hilum of spleen.
31/10	60	Tropical ulcer, gangrene.	Left lung: old fibrosed scar, no caseation of surrounding tubercles.
31/62	27	Pneumonia.	Right lung: old calcareous focus at apex.
32/91	50	——	Lungs: few sago-grain bodies. Caseous gland at hilum of spleen.
34/30	30	Cerebral abscess.	Right lung: upper lobe, small patch with caseous nodule in the centre and some surrounding fibrosis. Several pin-head, grey tubercles.
34/47	50	Tropical ulcer, pneumonia.	Right lung: apex, small contracted scar with puckering and some small grey surrounding tubercles. Left lung: old focus at apex with scarring and scattered small tubercles surrounding.
34/66	30	Transverse myelitis.	Old calcified nodule of one lung. (Site not noted.)
		Subjects with Lesions in Bronchial and/or Other Glands Without Obvious Lung Lesions.	
H/23	—	Pericarditis (non-tuberculous).	Bronchial glands: caseous points. Mesenteric glands: enlarged, show small dry caseous areas.
H/35	—	Tropical ulcer.	Bronchial glands: caseous foci present.
H/140	—	Dysentery.	Bronchial glands: small dry caseous foci.
H/156	18	Tropical ulcer.	Bronchial glands: many small caseous foci. Old caseo-calcareous nodule size of peppercorn just under visceral pleura. (Few old adhesions, apex of lung.)
H/184	30	"Malnutrition."	Bronchial glands: quiescent old caseous foci.
H/188	18	——	Bronchial glands: caseous foci.
H/231	20	Chronic nephritis.	Bronchial glands: caseous to suppurating. Hilar glands of spleen and liver enlarged, caseous, also glands in iliac region and along vertebral column. Few yellow tubercles in spleen.
A/270K	19	Dysentery.	Bronchial glands enlarged, caseous. Inguinal glands: suppurating. Mesenteric and pre-aortic glands caseous. No other lesions of tubercle.
A/271K	30	Pneumonia.	Enlarged caseous gland at bifurcation of trachea. No other tuberculous lesion seen.
A/182	14	Dysentery.	Bronchial and pretracheal glands enlarged, caseous. Z.N.[1]
T/361	21	Pneumonia.	Bronchial glands: enlarged, with tuberculous foci. Z.N.[1]
T/400	22	Intravenous administration of quinine.	Bronchial glands enlarged. Caseous foci present. Z.N.[1]
30/50	30	Hookworm anæmia.	Bronchial and pretracheal glands enlarged: caseous foci present.
30/17	23	Judicial hanging.	Cervical glands and gland at hilum of spleen showed caseous foci. Z.N.[1]
30/55	14	Drowning.	Old calcified bronchial gland of right side. No other signs of tuberculosis.
31/15	26	Beriberi.	Left lung: caseous foci hilar glands. Z.N.[1]

[1] Ziehl-Neelsen smear showed acid-fast bacilli.

entirely lacking; cases of some chronicity with fibrous tissue reaction did occur; retrogressive or quiescent lesions were occasionally seen: and tuberculin skin tests at that time and later (Heydon, 1937) gave evidence of an immunizing response to the increasing contact of these natives with European and Asiatic sources of infection.

During the period from 1922 to 1940 post-mortem examinations were performed on the bodies of 1541 natives from various districts of the mandated territory of New Guinea who died at Rabaul. Of these, tuberculosis was the chief cause of death in about 300. Unfortunately, some of the later records were lost during the war, and it was this loss that helped to discourage the writer from the task of analysing those records which survived, especially as it seemed to him in retrospect that they might have disclosed points essential to the argument he wished to develop.

The following figures include the 29 cases already reported by Heydon (1924). Of 225 bodies showing evidence of tuberculous infection, this disease was the chief cause of death in 182; it coexisted with other grave lesions in 13; while in 30 natives who had died from other causes, minor or obsolescent lesions of a tuberculous nature were recorded. These 225 natives comprised 195 males, mostly in the prime of life, 14 adult females, and 16 children up to the age of about twelve years. This uneven distribution is due to the circumstance that patients admitted to native hospitals were predominantly adult male, indentured, native labourers. Nevertheless, from its closer association with Europeans and Asiatics, this would be the group preeminently exposed to tuberculous infection in this area.

Summary of Autopsy Findings.

Adult Females.

Of the small group of 14 adult females, tuberculosis was the cause of death in 11. Minor foci, thought to be tuberculous in nature, but not bacteriologically confirmed, were seen in three women who died from other causes. The principal types were as follows: acute pulmonary types (one patient had caseous pneumonia, the other had lungs honeycombed with small cavities and studded with small caseous tubercles), two cases; extensive glandular and generalized tuberculous infection, six cases; the same, but with empyema and with miliary tubercles in the lungs, one case; caries of the lumbar part of the spine with miliary tuberculosis of the lungs, one case; tuberculosis of cervical, pretracheal, mediastinal and retro-peritoneal glands, caries of twelfth lumbar vertebra, one case; death due to other cause, minor tuberculous foci, three cases.

There was in this series no case of pulmonary cavitation of the type ordinarily seen in Europeans.

Children.

The ages of the children ranged from one year to about twelve years, and the types of the disease occurred roughly as follows: caseous bronchopneumonia, six cases; extensive tuberculous glands with meningitis, three cases; generalized miliary tuberculosis with meningitis, three cases; tuberculous glands with pericarditis, one case; glandular and generalized tuberculosis with peritonitis, one case; extensive intestinal tuberculous infection with massive involvement of abdominal glands, one case; glandular and renal tuberculosis with destruction of one kidney, one case. In one case, that of a child, aged seven years, there was, in addition to the glandular and meningeal involvement, a tuberculoma of the brain with internal hydrocephalus. Five of the children were from the Duke of York Islands, about twenty miles from Rabaul.

Adult and Adolescent Males.

The group of adult and adolescent males was made up of 152 subjects in whom infection by the tubercle bacillus was the cause of death, 11 in whom another grave disease, usually dysentery, coexisted with advanced tuberculosis and contributed to death, and 32 in which lesser, quiescent or even healed lesions were present in natives dead from other causes. Since these last-named subjects have a bearing on developed resistance, they are shown in some detail in Table I.

The 163 cases of advanced tuberculosis (with which are included the 11 natives dead of concurrent diseases) have been divided into two groups, as follows: (i) cases predominantly thoracic; (ii) cases in which extrathoracic lesions predominated. This follows the classification of the South African Tuberculosis Research Committee Report (1932). However, in further subdivision, the Melanesian cases appeared to fall more naturally into the following scheme.

The thoracic cases were as follows: (i) cases of acute caseating tuberculosis of the lungs, 49; (ii) cases in which similar pulmonary lesions were present, with the addition of gross lesions in the abdomen, 36; (iii) a group of cases in which lung lesions were less extensive, but widespread extrathoracic involvement was present, including the meninges in five and spinal caries in four, 17; (iv) a selected group of pulmonary cases in which some degree of fibrosis was recorded, 19. Because of the supposed bearing of these last-mentioned cases on the problem of the development of resistance, as indicated by attempts, however imperfect, to limit the direct spread of the infection, the principal lesions in this group are summarized in Table II.

The extrathoracic cases comprised 43 cases in which no pulmonary lesion other than miliary tuberculosis was recorded; they have been arranged as follows: (i) cases of predominantly glandular tuberculosis, usually with miliary sowing in the lungs and occasional lesions elsewhere, 11; (ii) cases in which gross serous membrane involvement was present (peritoneum 15, pericardium three), 18; (iii) cases in which meningeal involvement was a feature, with or without lesions included in (i) and (ii), 13; (iv) tuberculosis of the hip,[1] one.

Conclusion.

As far as these records go, they seem to support the views expressed by Heydon (1924) that in spite of the rapidly progressive form taken by the disease, in most cases there is evidence of a developing resistance in these natives. It is unfortunate that some of the records of the later years were lost, since these contained a few cases of pulmonary cavitation in which the fibrosis was considerable, but which cannot now be cited in detail. Whether twenty years is a sufficient period for this supposed change in the pathological picture is a question the writer must leave for others to debate.

[1] Although caries of the spine (with psoas abscess in three instances) was seen in nine bodies in which there were other gross lesions of the tubercle, this was the only case of a tuberculous joint without apparent infection by the bacillus elsewhere in the body. It was recorded by Dr. F. Trennery at Rabaul in 1929.

TABLE II.

Subjects with Some Degree of Fibrous Reaction to Pulmonary Tuberculosis.

Reference Number.	Age in Years.	Brief Description of the Lesions.[1]
A/129	32	Right lung: extensive cavitation.
A/135	20	Lungs: extensive consolidation and cavitation; some well-formed but not very thick-walled cavities. Bronchial glands caseous. Cervical glands grossly tuberculous.
A/144	30	Left lung: some rather thick-walled cavities, elsewhere nodules with softened centres, miliary and confluent areas. Intestinal ulcers and mesenteric gland involvement.
A/162	40	Lungs: extensive honeycombing with imperfect fibrosis. Left lung with a distinct zone of fibrous tissue tending to wall off apical portion. Tuberculous ulceration of larynx.
A/180	21	Lungs: large loculated cavities with definite fibrous tissue. Cavities on left side smaller and with thinner walls. Laryngeal tuberculosis.
A/183	27	Lungs: crepe-rubber consistency and appearance due to many pea-sized cavities and interstitial fibrosis, also areas of miliary and confluent tuberculosis. Abdominal glands suppurating.
A/184	26	Right lung: large cavity with few fibrous bands. Rest of lung, confluent tubercles and fibrous tissue. Left lung: cavities size of walnut with moderately thick walls and consolidated areas. Bronchial glands enlarged and indurated, not caseous. Intestinal ulcers, mesenteric glands infiltrated.
A/202	25	Lungs: miliary and confluent tubercles with small cavities and fibrous reaction.
A/203	25	Lungs: confluent tubercles and relatively thick-walled cavities. Left lung, smaller thinner-walled cavities than right. Intestinal ulcers and grossly invaded mesenteric glands.
A/206	27	Miliary and confluent tubercles; cavity 1·5 inches in diameter left upper lobe, well marked fibrous wall, containing blood clot.
30/19	18	Left lung: large loculated cavity with distinct fibrous wall occupying almost whole upper lobe. Right lung: more recent smaller cavities with little fibrosis. Intestinal ulceration.
30/36	18	Lungs: extensive caseous lesions and excavation; some fibrous reaction. Right upper lobe with some almost cartilaginous fibrous tissue. Bronchial and pretracheal glands caseous.
30/48	25	Right lung: caseation and cavitation; some fibrous tissue reaction. Left lung: thin-walled cavity 1·5 inches in diameter, remainder of lung invaded by tubercles in various stages. Intestinal ulcers and great enlargement of mesenteric glands.
31/2	28	Right lung: lower lobe with large yellow-grey caseous consolidation, grey tubercles on pleura. No general sowing in rest of lung. Left lung: whole lower lobe a ragged cavity crossed by bridles. No general extension to rest of lung.
31/38	22	Lungs: small blood-filled cavities, upper lobes with some surrounding fibrous reaction. Numerous scattered miliary tubercles. Kidneys, few small tubercles.
33/23	30	Right lung: consolidated (resembling pneumococcal hepatization). Left lung: cavity 1·5 inches in diameter in upper lobe, rather thick-walled; elsewhere in lobe confluent tubercles; lower lobe gangrenous.
34/54	28	Right lung: whole upper lobe occupied by large cavity divided by fibrous band. Rest of lung infiltrated. Left lung densely infiltrated with tubercles, no cavitation. Bronchial glands caseous, but no general spread.
34/56	23	Lungs: cavities with hæmorrhage both upper lobes, some limiting fibrous reaction, but extensive spread in lower lobes. Bronchial glands caseous.
34/58	25	Lungs: right, loculated cavity lower third of upper lobe. Extensive infiltration with confluent yellow tubercles. Some fibrosis about cavity. Bronchial glands caseous. Intestinal ulceration and mesenteric gland involvement.

[1] No special mention has been made of pleural adhesions, which were more or less universally present and are common in natives apart from tuberculous infection.

Acknowledgements.

The writer has supplemented the notes made by himself with those of several others who performed post-mortem examinations at Rabaul and made their notes available—notably Dr. G. A. M. Heydon, Dr. A. H. Baldwin, and Dr. F. Trennery, to all of whom he expresses belated thanks. To Dr. G. Bruce White, who read the manuscript and made helpful comment, he is also grateful.

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DEFECTIVE BLOOD COAGULATION ASSOCIATED WITH INTRAUTERINE FŒTAL DEATH.

By J. M. FARRAR,

Sydney.

IN recent years increasing attention has been paid to disordered coagulation of the blood as a cause of hæmorrhage in pregnancy, labour and the puerperium. It is now well established that moderate or severe concealed accidental hæmorrhage, long-standing intrauterine fœtal death, amniotic fluid embolism and tumultuous labour are possible precursors of a clotting defect. Defective blood coagulation has been reported in patients with missed abortion, and following the inhalation of vomitus after anæsthesia (Barry *et alii*, 1955). Scott (1955) mentions that the condition may occur in patients with hydatidiform mole. O'Driscoll and Lavelle (1955) state that defective coagulation has been reported in toxæmia of pregnancy and as a cause of delayed post-partum hæmorrhage.

The coagulation defect in the above conditions may be due to various abnormalities, but the most important are diminution in amount or absence of the blood fibrinogen (hypofibrinogenæmia or afibrinogenæmia) or the presence of an active fibrinolysin. Decrease in the amount of prothrombin, antihæmophiliac globulin, Factor V, Factor VII and platelets and increased thrombin-inhibitory activity have at times been reported, but in all instances they were of secondary importance in causing the bleeding.

Hypofibrinogenæmia and fibrinolysis may occur separately or together. Fibrinolysis may be a factor in the production of hypofibrinogenæmia, and the cause of hypofibrinogenæmia may also play a part in the production of fibrinolysins. With this complexity it is small wonder that at times we can only guess at the mechanism of production of the coagulation defect.

HYPOFIBRINOGENÆMIA AND FIBRINOLYSIS IN OBSTETRICS.

Hypofibrinogenæmia.

Hypofibrinogenæmia in most instances is probably due to excessive utilization of the circulating fibrinogen or to its destruction by fibrinolysin. When dextran is used as a plasma substitute, fibrinogen may be inactivated by the formation of a dextran-fibrinogen complex (Scott, 1955).

Excessive utilization of fibrinogen may occur in the presence of severe hæmorrhage associated with fibrinolysis because the clots are immediately dissolved. Fibrinogen levels in the blood may be low in the absence of hæmorrhage when the circulation is flooded with material rich in thromboplastin (*liquor amnii* or products of placental degeneration or damage), because coagulation is initiated and a film of fibrin is deposited on the vascular endothelium. (This is usually lysed rapidly, since the fibrinolytic mechanism of the blood is likely to be activated.) The production of disseminated fibrin embolism uses up large quantities of circulating fibrinogen; and when a critical level is passed obvious hæmorrhagic phenomena are liable to occur. (The critical level is estimated from various reports at 100 to 200 milligrammes per 100 millilitres; normal levels in late pregnancy and labour are reported as 450 to 600 milligrammes per 100 millilitres.)

It has been estimated that the total quantity of circulating fibrinogen in the body amounts to eight to twelve grammes, and that a 500-millilitre blood transfusion may increase the level of fibrinogen by 10 to 20 milligrammes per 100 millilitres. The liver can replace the lost fibrinogen rapidly, and in the less severe cases the fibrinogen may be kept above the critical level by regeneration with or without blood transfusion. However, in the more severe cases this may be impossible, and it is here that the intravenous administration of fibrinogen may be life-saving.

Fibrinolysis.

Obscurity still surrounds many aspects of the phenomenon by which enzymes destroy formed fibrin. Experimentally fibrinolytic activity in blood may be induced *in vitro* or in animals. Clinically it has been observed after various forms of stress, either mental or physical; it has been noted frequently in obstetrics, and after operations on the lungs. It may be seen *post mortem*, especially in cases of sudden death. It is not yet clear whether the actual process of fibrin destruction is due to the action of the same lytic factor under these diverse conditions.

Biggs and Macfarlane (1953) have described four varieties of fibrinolysin:

1. The enzymes producing fibrinolysis *in vitro*: (a) Plasmin, which, together with its precursor plasminogen, occurs in the globulin fraction of the plasma. Antiplasmin is associated with the albumin fraction. Plasmin destroys fibrinogen as well as fibrin. Plasminogen is activated by bacterial extracts such as streptokinase. Antiplasmin is destroyed by chloroform. (b) A similar enzyme which may be activated by tissue extracts containing fibrinokinase. Differences may be detected between this enzyme and plasmin on laboratory examination.

2. A fibrinolysin which appears after severe exercise or emotional disturbance. It differs from plasmin in that it does not attack fibrinogen.

3. The enzyme responsible for post-mortem fibrinolysis. This has many features in common with those observed after severe exercise.

Little work has been done to determine the nature of the fibrinolysins encountered in obstetrics. Soulier *et alii* (1952) report the case of a patient who died after criminal abortion: shock and incoagulability of the blood occurred during laparotomy for suspected uterine perforation. He concluded that the lysin was plasmin.

To explain fibrinolysis in pregnancy several mechanisms may be postulated. It is possible that large quantities of fibrinolytic enzyme may be released into the circulation directly from the placenta or from other parts of the uterus or its contents. On the other hand, the normal plasminogen-plasmin-antiplasmin system may be activated by: (i) some product of placental damage or degeneration released into the circulation; (ii) the stimulus of widespread injury to the vascular endothelium following disseminated fibrin embolism; (iii) shock which often precedes the conditions known to produce fibrinolysis.

Treatment aims to maintain the blood fibrinogen above the initial level.

Figure I shows diagrammatically some of the processes which may result in the formation of hypofibrinogenæmia or fibrinolysis in obstetrics.

COAGULATION DEFECT WITH DEATH IN UTERO.

Intrauterine fœtal death assumes a new importance now that it is recognized that it may be associated with impaired coagulation of the blood.

Clinical Features.

A coagulation defect following fœtal death *in utero* is not common. In the reported cases the retention of the dead fœtus in the uterus has been of long standing—that is, more than four weeks. On the other hand, duration of retention of the dead fœtus in the uterus does not appear to be the sole determining factor; Barry *et alii* (1955) report two cases each associated with eight weeks' retention of a dead fœtus in the uterus, but in only one of them did hæmorrhagic phenomena occur.

In most of the reported cases the fœtal death has been attributed to Rh incompatibility, yet this is not essential, since Reid *et alii* (1953) reported two cases in Rh-positive women with Rh-positive husbands. In one of these, investigations failed to reveal any evidence of A B O incompatibility; in the second no mention was made of such studies, although the patient belonged to Group O and the husband to Group B. Frick and McKelvey (1955) report an instance in which both the patient and her husband were of identical genotype.

The first suspicion of abnormality may be intra-partum or post-partum hæmorrhage. Usually, however, hæmorrhagic symptoms precede blood loss from the uterus, and these may include a generalized purpuric eruption, ecchymoses at injection sites, or bleeding from the gums or other mucous surfaces.

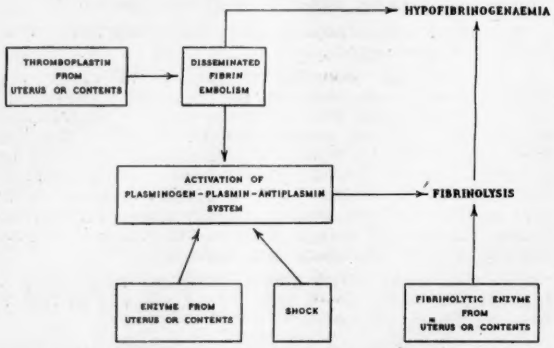

FIGURE I.

Nature of the Coagulation Defect.

The most important abnormalities in the coagulation mechanism following fœtal death *in utero* are hypofibrinogenæmia or fibrinolysis. One or the other or a combination of the two has been reported in every instance, and decreases in other clotting factors (prothrombin, anti-hæmophiliac globulin, Factor V, Factor VII, platelets) or increased plasma thrombin-inhibitory activity have always been considered of secondary importance as a cause of the bleeding.

The placenta is considered the most likely source of the substances initiating the disordered coagulation mechanism, and they may be forced into the circulation by uterine contractions before or during labour. Reid *et alii* (1953) believe that the degree of placental autolysis is an important factor determining whether a clotting defect will occur in any particular patient. They consider the condition particularly likely to occur in association with a placenta autolysed to the point of almost complete liquefaction.

Diagnosis.

While not a common occurrence in long-standing intra-uterine fœtal death, the possibility of coagulation defect should always be remembered. Before the onset of labour the condition has to be distinguished from other blood dyscrasias. During labour it must be decided whether or not bleeding from the uterus is an uncomplicated ante-partum hæmorrhage. *Post partum* the diagnosis should be one of exclusion, and the much more common atonic or traumatic causes of post-partum hæmorrhage must be ruled out first.

Ideally the onset of obvious bleeding should be anticipated by periodic quantitative determination of the blood fibrinogen level and fibrinolysin concentration. This is usually possible if the patient is seen before labour has commenced.

However, when one is confronted by unexpected and severe bleeding, rapid assessment must be made of the part played by hypofibrinogenæmia or fibrinolysis in its production. Suspicion may be aroused by the fact that the blood lost fails to clot or redissolves on clotting, or by the fact that bleeding is occurring from parts other than the uterus, such as the mouth or the sites of needle punctures. Confirmation may be obtained at the bedside by various tests; these have been described by Schneider (1952), Reid *et alii* (1953) and Scott (1955). A modification of Scott's test was used in the case described below. One millilitre of a solution of freshly prepared thrombin (1000 N.I.H. units per millilitre) was added to one millilitre of citrated blood in a small test tube, and the speed of the clot formation and the stability of the clot were observed.

Management.

The management of a patient in whom intrauterine fœtal death occurs may be considered under two main headings: (*a*) before delivery, (*b*) after delivery.

Before Delivery.

Before Occurrence of Hypofibrinogenæmia or Fibrinolysis. Although opinion varies, I believe that the most logical course of management before the occurrence of hypofibrinogenæmia or fibrinolysis is induction of labour shortly after fœtal death has been verified. This is most safely attempted by the use of "Pitocin" by drip administration, with strict avoidance of any intravaginal manipulation. Induction by this means, repeated if necessary, should be successful in a very large percentage of cases. Few patients should remain to return a dead fœtus for longer than four weeks. Attempts to induce labour after intrauterine fœtal death of more than four weeks' duration are potentially dangerous and should be avoided. "Pitocin" will cause uterine contractions but may fail to start labour. The result will be increased likelihood of absorption of products of placental autolysis and hence of the development of coagulation defect, without bringing the termination of pregnancy any closer.

After Occurrence of Hypofibrinogenæmia and Fibrinolysis. Once there is evidence of hypofibrinogenæmia and fibrinolysis, fibrinogen should be replaced and blood loss corrected by blood transfusion. The problem of removal of the source of fibrinolysins may present the utmost difficulty. Though one is always reluctant to perform a section on a patient in whom fœtal death has occurred *in utero*, this may rarely be necessary. It must be emphasized that section before the correction of hypofibrinogenæmia is potentially lethal. The result is an increase in the area from which uncontrollable bleeding will occur.

After Delivery.

After delivery all that should be required is correction of the hypofibrinogenæmia and restoration of the blood lost. The circulation will not now be flooded with additional fibrinolysins, and energy must be directed towards enabling clot formation to keep pace with clot lysis until the activity of the fibrinolysin already circulating has disappeared.

Hysterectomy will merely transfer the site of the bleeding from the uterine sinuses to the pelvic vessels and the abdominal wall.

REPORT OF CASE.

A woman, aged twenty-seven years, was first seen in the eighth week of her fourth pregnancy. Her blood group was A, Rh-negative. Her husband's blood group was O, Rh-positive.

Investigation of her previous obstetric history revealed that her first pregnancy had occurred in 1949. She miscarried at three months, the cause being unknown. The second pregnancy in 1950 ended with the delivery at term of a macerated fœtus which had been dead for one month. No information was available about blood loss. No examination was made for the presence of agglutinins. The third pregnancy occurred in 1952, with delivery at term of a macerated fœtus which had been dead for one month. Blood loss was 40 ounces associated with a retained placenta. No maternal anti-Rh agglutinins were detected, but the autopsy findings suggested that the death had been caused by hæmolytic disease of the newborn.

In the present pregnancy, at the sixteenth week, anti-D agglutinins (the titre in albumin was 1:256) were detected in the serum. Fœtal movements ceased during the twenty-third week with the fundus one to two fingers' breadth above the umbilicus. After thirty-four weeks' amenorrhœa, and with intrauterine retention of a dead fœtus for eleven weeks, the fundus was just below the umbilicus. Labour was induced at this stage by "Pitocin" given by drip administration.

Contractions had been present for three hours when a moderate loss of blood occurred which did not clot from the vagina. Blood was collected from the patient and tested by the method described above. An unstable clot was formed, which redissolved in fifteen minutes (Figure II, tube 1).

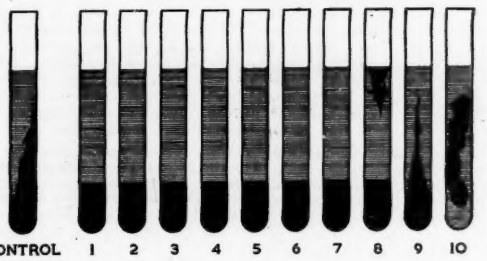

FIGURE II.

It was decided that fibrinolysis and possibly hypofibrinogenæmia were present.

Blood was taken for more detailed laboratory investigations with the following results: The coagulation time (by Lee and White's method) was twelve minutes; a poor clot formed, which redissolved in sixteen minutes. The platelet count was 240,000 per cubic millimetre. The prothrombin time was twenty-three seconds (control, 23 seconds). The prothrombin consumption time was normal. Fibrinolysin (as determined by Macfarlane's plasma dilution method) was present. Increased plasma thrombin-inhibitory activity was not detected.

An increased rate of blood loss followed the expulsion of the fœtus, intact sac and placenta, one and a half hours after the initial hæmorrhage. In spite of the intravenous injection of ergometrine the uterus would not remain firmly contracted unless continuous abdominal massage was performed. By combining massage with pressure of the fundus against the sacral promontory it was possible to retard the rate of blood loss. An intravenous injection of fibrinogen (three grammes) in normal saline was given while a blood transfusion was begun. A further injection of fibrinogen (one gramme) was given one hour after delivery.

Compared with the findings of the test made at the onset of bleeding, the rate of dissolution of clot remained unchanged just before delivery (tube 2), shortly after the first injection of fibrinogen (tube 3), one hour after the second injection of fibrinogen (tube 4) and three hours later (tube 5). At the time the last test was taken the vaginal blood loss was negligible and the fundus was firmly contracted.

The sixth test for fibrinolysis was carried out two hours after the fifth, and repeated examinations were made every two hours. Tubes 6 and 7 still showed complete lysis of clot, yet the time taken for this to occur was increased. Ten hours after delivery (tube 8) the clot did not dissolve completely, yet it was not until fourteen hours after delivery (tube 10) that a firm clot was formed which did not dissolve.

The total blood loss was estimated at two litres. Two and a half litres of blood (containing an estimated 4·5 grammes of fibrinogen) and four grammes of fibrinogen were given. At no time was the patient shocked. The blood pressure never fell below 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. Dextran was not administered at any stage. The puerperium was uneventful and the patient was discharged from hospital on the seventh day with a hæmoglobin concentration of 15 grammes *per centum*.

Discussion.

Fibrinolysis was an outstanding feature of the coagulation defect in this patient. The estimation of fibrinogen was not made, so that, although theoretically the blood fibrinogen level should have been lowered, there is no proof that his was so. No decrease in clotting factors other than fibrinogen was detected. There was no increase in plasma thrombin-inhibitory activity.

The placenta was firm and its gross and microscopic structure was quite well preserved, which provides evidence that autolysis to the point of liquefaction is not essential for the development of this type of coagulation defect.

The disordered blood coagulation, then, was characterized by the presence of an active fibrinolysin, probably with accompanying hypofibrinogenæmia. Shock was not responsible for the genesis of the fibrinolysin, and circumstances indicated that a lysin or a lysin-activator was dispersed into the circulation from the uterus, since bleeding was first noted three hours after uterine contractions began. Generalized hæmorrhagic phenomena did not occur, and we can only guess whether these would have appeared later had artificial termination of the pregnancy not been performed.

Induction of labour was unwise so long after intrauterine fœtal death, and it was fortunate that delivery occurred so soon after the bleeding began, since severe generalized hæmorrhage may have occurred had delivery been delayed. In this event it would have been difficult to get rid of the source of the lysins; section would have been hazardous in the presence of coagulation defect, and oxytocics might be expected to have increased the outpouring of lysins from the uterus.

It is interesting to note that the bleeding was controlled clinically while there was still in-vitro evidence of fibrinolysis. It may be wondered what part, if any, the administered fibrinogen played in the cessation of the hæmorrhage. Two factors are essential to control hæmorrhage of any sort: a mechanical one of vasoconstriction, and the coagulation of the blood. In the early stages the uterine mechanism for control of hæmorrhage was not acting to the fullest extent; it is probable that the blood fibringen was below the critical level and fibrinolysis was present. Later the uterus remained firmly contracted, and it is probable that the fibrinogen level had risen; so that now clot formation could keep pace with clot lysis. A contributing factor to the early cessation of bleeding may have been the fact that, as only a small fœtus was passed, there was little likelihood of tears which could not be controlled by contraction of the myometrium.

SUMMARY.

1. The conditions in obstetric practice which may be associated with defective blood coagulation are considered. It is pointed out that the most common and most important abnormalities detected in the blood in such cases are hypofibrinogenæmia or afibrinogenæmia and/or fibrinolysis.

2. Possible mechanisms of production of those abnormalities when encountered in obstetrics are discussed.

3. Consideration is given to mechanisms of production of the coagulation defect associated with long-standing intrauterine fœtal death. Mention is made of clinical features, diagnosis and management.

4. A case is reported of coagulation defect associated with long-standing retention of a dead fœtus *in utero*.

ACKNOWLEDGEMENTS.

A large part of the management in this case was carried out by Dr. R. P. Shearman, Dr. W. G. Grigor and the resident medical staff of King George V Memorial Hospital. I am indebted to Dr. C. S. H. Reed and Dr. B. G. Firkin,

of the Clinical Research Unit, Royal Prince Alfred Hospital, for advice and for carrying out the laboratory investigations.

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Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Antibiotics Annual, 1955-1956", edited by Henry Welch, Ph.D., and Félix Martí-Ibáñez, M.D.; 1956. New York: Medical Encyclopedia, Incorporated. 10" × 7", pp. 1010, with illustrations. Price: $10.00.

There are more than 150 chapter headings, some by more than one author.

"A Manual of Anæsthetic Techniques", by William J. Pryor, M.B., Ch.B. (N.Z.), F.F.A.R.C.S. (Eng.), D.A. (Eng.), M.F.A., R.A.C.S., with foreword by J. H. T. Challis, M.R.C.S. (Eng.), L.R.C.P. (Lond), F.F.A.R.C.S. (Eng.), D.A. (Eng.); 1956. Bristol: John Wright and Sons, Limited. 5¼" × 8¼", pp. 234, with illustrations. Price: 27s. 6d.

Written for house surgeons and registrars commencing the practice of anæsthetics.

"Gestation", edited by Claude A. Villee, Ph.D.; 1956. New York: The Josiah Macy, Jr. Foundation Publications. 9" × 6", pp. 248, with illustrations. Price: $5.00.

There were 13 participants in the conference and some guests. Seven subjects were discussed, dealing chiefly with gestation in mammals.

"Pulmonary Circulation and Respiratory Function: A Symposium Held at Queen's College, Dundee"; 1956. Edinburgh and London: E. and S. Livingstone, Limited. 7¼" × 9¾", pp. 54, with illustrations. Price: 12s. 6d.

The discussions extended over two days and seven subjects were dealt with on each.

"History of the Second World War: United Kingdom Medical Series", Editor-in-Chief, Sir Arthur S. MacNalty, K.C.B., M.A., M.D., F.R.C.P., F.R.C.S. "The Royal Naval Medical Service", by Surgeon Captain J. L. S. Coulter, D.S.C., R.N. Volume II: Operations; 1956. London: Her Majesty's Stationery Office 9¼" × 6", pp. 543, with 19 illustrations. Price: £2 17s. 6d.

This volume completes the volumes of the "History" which deal with the Royal Navy.

"Basic Otolaryngology", by Francis L. Lederer, B.Sc., M.D., F.A.C.S., F.I.C.S. (Hon.), and Abraham R. Hollender, M.Sc., M.D., F.A.C.S.; Fourth Edition; 1956 Philadelphia: F. A. Davis Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 453, with many illustrations. Price: £3 14s. 3d.

A book for students.

"The Year Book of Urology (1955-1956 Year Book Series), edited by William Wallace Scott, M.D., Ph.D.; 1956. Chicago: The Year Book Publishers, Incorporated. 5" × 7½", pp. 398, with many illustrations. Price: $6.50.

One of the Practical Medicine Series of Year Books.

"The Year Book of the Eye, Ear, Nose and Throat (1955-1956 Year Book Series)", edited by Derrick Vail, B.A., M.D., D.Oph. (Oxon.), F.A.C.S., F.R.C.S. (Hon.), and John R. Lindsay, M.D.; 1956. Chicago: The Year Book Publishers, Incorporated 5" × 7½", pp. 471, with many illustrations. Price: $6.50.

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"Pediatric Clinics of North America: Symposium on Pediatric Proctology"; 1956. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 5½" × 9", pp. 228, with illustrations. Price: £6 15s. *per annum*.

Consists of fourteen chapters and an introduction; there are 22 contributors. Anatomy, anæsthesia and pathological conditions are considered.

"Anesthesia, Thief of Pain", by Sylvan M. Shane, with introduction by Henry E. Sigerist; 1956. New York: Vantage Press. 5½" × 8", pp. 87, with illustrations. Price: $2.50.

A book for the general public.

"A Manual of Obstetrics", by the late O'Donel Browne, M.B., M.A.O., M.A., Litt.D., F.R.C.P.I., F.R.C.O.G., edited and largely rewritten by J. G. Gallagher, M.D., M.A.O., F.R.C.P.I., M.R.C.O.G.; 1956. Bristol: John Wright and Sons, Limited. 9" × 5½", pp. 274, with 205 illustrations. Price: 37s. 6d.

The first edition was published in 1936; it is intended for the student and practitioner.

"Progress in Neurobiology: I. Neurochemistry", edited by Saul R. Korey, M.D., and John I. Nurnberger, M.D., with 23 contributors; 1956. New York: Hoeber-Harper, Incorporated. 9" × 6", pp. 225, with illustrations. Price: $6.75.

There are sixteen chapters and twenty-four contributors.

"A Manual of Fractures and Dislocations", by Barbara Bartlett Stimson, A.B., M.D., Med.Sc.D., F.A.C.S.; Third Edition; 1956. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 5¼" × 7¼", pp. 224, with illustrations. Price: £2 9s. 6d.

Intended for students, internes and general practitioners.

"Peripheral Vascular Disorders", edited by Peter Martin, V.R.D., M.Chir., F.R.C.S.Ed., R. Beverley Lynn, M.D., F.R.C.S., J. Henry Dible, M.B., LL.D., F.R.C.P., and Ian Aird, Ch.M., F.R.C.S.; 1956. Edinburgh and London: E. and S. Livingstone, Limited. 10" × 7", pp. 855, with many illustrations. Price: £5 10s.

The number of authors is nine. Anatomy and physiology are covered. The description of pathology of obliterative disease is based on specimens collected over a period of sixteen years.

"Emotional Problems of Early Childhood", edited by Gerald Caplan, M.D.; 1955. London: Tavistock Publications, Limited. 9¼" × 6¼", pp. 558. Price: £2 2s.

Based on papers presented to the International Institute of Child Psychiatry at Toronto in August, 1954.

"The Facts of Mental Health and Illness", by K. R. Stallworthy, M.B., Ch.B., D.P.M.; 1956. Christchurch: N. M. Peryer, Limited. 8¼" × 5¼", pp. 215. Price: 18s. 6d.

A book for the layman "who has an interest in the mind and the way it works, in health and illness".

"Babies Growing Up: Their Progress from Before Birth Right Through the Nursery Years", by Nurse McKay. London: Routledge and Kegan Paul, Limited. Sydney: Walter Standish and Sons. 7¼" × 4¾", pp. 239. Price: 17s. 6d.

A parents' manual.

The Medical Journal of Australia

SATURDAY, JULY 14, 1956.

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SURGICAL PHYSIOLOGY.

DR. WARREN H. COLE, in his presidential address to the Clinical Congress of the American College of Surgeons in Chicago on November 4, 1955, drew attention to the fact that the mortality rate for subtotal gastrectomy was 25% to 30% in the hands of Lord Moynihan, Will Mayo, John Deaver and other surgeons of high standing, whereas now it is 3% to 5%. Is the surgeon of today a superior being? Certainly not. Has the operative technique improved? No, it has not. "What then is the explanation of this sharp reduction to 20 to 25 per cent. of the original figure? The answer is very simple: we have learned to appreciate some of the ordinary features of surgical physiology, namely, that we must not do major operations on patients with dehydration, electrolyte deficiency, anæmia and malnutrition until corrective measures have been utilized. Years ago patients with carcinoma of the stomach were sent to the operating room without any pre-operative preparation except for administration of a generous amount of a saline purge which unfortunately added to the patient's dehydration, electrolyte imbalance and malnutrition." In the earlier decades of this century one of Australia's most distinguished and most original surgeons repeatedly warned his students not to "import into the operating theatre the lore of the physiological laboratory". To him and to his generation of surgeons anatomy was the all-important basic subject to be learned with thoroughness; the function of any organ could be explained in a few lines at the conclusion of the full morphological description in text-books of anatomy. There was only one place in which to learn surgery, namely, the hospital with its surgical wards and operating theatre. Physiology might be useful to the physician, but to the surgeon it had no value, sometimes even the reverse. As Warren Cole has stated, a change has come over the scene; today, he remarked, a patient about to have a major operation, especially in the intestinal tract, will have his weight and calorie intake carefully checked, and if he is incapable of digesting enough food to make up for the depletion which has preceded his appeal to the surgeon, nutriment will be administered by the intravenous injection of nutrient fluids or by blood transfusion.

The distribution of emphasis between the clinical on the one hand and the pre-clinical and basic on the other has given rise to much controversy in the past and will continue to be a bone of contention. It would all be so simple if medical science, in the broadest sense of the term, was static; then only those chapters of chemistry, physics, biology and physiology directly applicable to clinical practice would need to be taught; but we must face the fact that medical science is progressive and is continually borrowing, and often paying back with high interest, principles and techniques formerly regarded as remote from what is necessary in the activities of the physician and surgeon. When Röntgen in 1895 announced the discovery of the rays which bear his name, rays which could pass through soft tissues but make bones throw a shadow, the members of the medical profession at once realized that a wonderful weapon had been forged to help in the fight against disease and accident, but few were able to set up the apparatus or understand the principles on which it was based; they remembered something about the parallelogram of forces, Atwood's machine, the path of a projectile, the Gregorian telescope and such like, but the bombardment of an anode by a stream of negative electrons was something beyond their ken, and so those interested had to consult physicists and once more learn, after a long vacation, a little natural philosophy.

To take sides in the controversy whether clinical observation has contributed more than laboratory experiment is an idle contest; there have been, and there will always be, give and take. The coagulation of the blood is of high importance in surgery, but to our knowledge of the complex chain of reactions which brings this about clinical medicine and surgery have contributed nothing at all; on the other hand, the action of rubella on the pregnant woman, a consideration of major importance to human welfare, has been studied not in the laboratory but at the bedside. Anæsthesia was the first non-surgical activity to be admitted into the operating theatre. Up to a few decades ago the successful surgeon liked to give a helping hand to a struggling junior and let him earn a guinea as an anæsthetist. Today no surgeon, unless in dire emergency, would entrust anæsthesia to anyone except a trained anæsthetist. More and more are the services of the bacteriologist and X-ray expert called into requisition. Formerly the only interest the surgeon took in the diet of his patient was that it should not induce vomiting during the operation; now we know that the physical state of the blood and of the protein resources of the tissues are of high importance. Especially in America do we find the opinion expressed that a spell of research during residentship can be helpful. To quote Warren Cole again: "It teaches the young surgeon a lot

of fundamental facts about physiology and numerous other basic sciences; likewise it encourages him to apply scientific methods in solving clinical problems and should also instil a scientific curiosity of a lasting type in the mind and soul of the surgeon." Grumbles are constantly being heard and will continue to be heard amongst practising surgeons that the medical curriculum contains studies that can never be of use in the subsequent careers of the students. Such critics should be careful; they may have to eat their own words a little later.

Current Comment.

PRIMIDONE IN EPILEPSY.

PRIMIDONE was first introduced into experimental neurological practice in 1950 under the now familiar proprietary name of "Mysoline". A preliminary trial of the drug in epileptic patients was made by G. E. Smythe, and after favourable reports of this, a larger trial was instituted and the report was published by R. Handley and A. S. R. Stewart.[1]

Forty patients in an epileptic colony who were continuing to have *grand mal* seizures despite conventional therapy were treated by primidone in doses by mouth of up to 1·6 grammes daily. No toxic effects were noted when the new treatment gradually replaced the old. It was found that the control groups quickly recognized those who were receiving treatment by the obvious improvement in their condition. Attacks were reduced in 80% of the patients and in 30% were abolished altogether. Further clinical trials followed and B. H. Smith and F. L. McNaughton[2] were able to report that primidone appeared to give beneficial effects in all types of epilepsy. However, subsequent reports have not upheld the general usefulness of the drug in all types of epilepsy.

C. W. M. Whitty,[3] in a report of the use of primidone in the treatment of 70 previously uncontrolled epileptics, found that there were no serious toxic effects, though drowsiness occurred in nearly half the patients and some developed a mild confusional psychosis. Regardless of the type of epilepsy, forty-nine patients improved with primidone and this included several in whom the attacks were of psychomotor type. Each in a separate report, B. G. Burton-Bradley, J. A. Game and G. Selby[4] found that at least half of the refractory epileptics were benefited by primidone therapy. Selby noted that the drug was of particular value in *grand mal* epilepsy. No serious toxic effects were reported by any of these authors. J. B. Lyons and L. A. Liversedge[5] reported on the use of primidone in out-patients. Used alone, the drug produced a 75% or greater reduction in the number of attacks in 21 out of 42 patients. Side effects such as drowsiness tended to disappear after two or three weeks of treatment. Some patients with focal disturbance of the electroencephalograph recording appeared to develop a more normal electrical pattern. E. S. Sharpe[6] used primidone on mentally defective epileptics and reported favourable results with *grand mal* epilepsy but not in cases of *petit mal*. Opinion has probably tended to reject the use of primidone in *petit mal*, though the value of the drug for patients with *grand mal* is without doubt.

S. Livingston and D. Peterson[7] have now reported on the results of using primidone in a large group of epileptic patients over a long period of time. In the survey were 486 patients of whom 294 were resistant to previous therapy and 192 had not formally received treatment. The duration of therapy ranged from six months to three years and the dosage from 375 milligrammes to 2000 milligrammes daily in two to four doses. Of the 311 patients with pure major or motor seizures, 57% of the previously untreated, 28% of the previously mildly refractory and 17% of the previously completely refractory patients were completely controlled by primidone. Among the patients with other types of epilepsy, no significant benefit was forthcoming. Primidone caused such severe drowsiness, dizziness or ataxia in 46 patients that the treatment was withdrawn. Otherwise, no side effects were of significance. Two patients developed morbilliform skin rashes which did not recur when treatment was recommenced. No blood or urinary abnormalities were noticed throughout the series. A conclusion to be drawn is that primidone is a safe and effective drug for the routine treatment of major motor epilepsy.

[1] *Lancet*, April 12, 1952.
[2] *Canad. M. A. J.*, May, 1953.
[3] *Brit. M. J.*, September 5, 1953.
[4] M. J. AUSTRALIA, November 7, 1953.
[5] *Brit. M. J.*, September 11, 1954.
[6] *Brit. M. J.*, September 11, 1954.
[7] *New England J. Med.*, February 16, 1956.

TUBERCULOUS MILIARY NECROSIS WITH PANCYTOPENIA.

THE term pancytopenia describes a reduction in the red cells, the leucocytes and the blood platelets. The leucopenia is usually due to an absolute reduction in the granular cells, but in some cases the lymphocytes are reduced in number as well. In the past these cases have often been described as "aplastic" or "refractory anæmia", but pancytopenia is a more general term, and is useful because it is purely descriptive and free from any implied preconceptions as to ætiology. This condition of the blood occurs in many diseases, notably in leuchæmia and in other reticuloendothelioses; it may be caused by chemical agents such as benzol, gold salts, volatile insecticides, hair dyes, and by physical agents such as ioninzing radiation. The degree in which each of the formed elements of the blood is affected may vary, and the malady may be sudden and rapidly fatal, or slow and insidious. There is no doubt that intermediate and transitional forms occur not only in the primary diseases—leuchæmia, myelosclerosis and idiopathic pancytopenia—but also in the blood conditions caused by chemical and physical agencies.

With so many possibilities as to ætiology it is most important that every case of pancytopenia should be fully investigated, not only from the point of view of treatment, but for the furtherance of knowledge and the prevention of disease. The term "idiopathic" must be used only as a last resort. A recent article by W. E. Medd and F. G. H. Hayhoe[1] lends force to this argument. They describe four patients in whom pancytopenia in the peripheral blood was associated with miliary tuberculosis. In one of these cases a diagnosis was made during life by aspiration liver biopsy, and the patient appears to have been satisfactorily treated with streptomycin and "Isoniazid" up to the time of writing 18 months later. In a second case splenectomy was performed, and histological examination of the spleen provided the diagnosis, but the patient died twenty-two days later. The other two patients were regarded as suffering from primary blood dyscrasia during life and the true diagnosis was made only *post mortem*. Medd and Hayhoe point out that "the hæmatological complications of tuberculosis fall loosely into three groups: leukæmoid reactions, myelosclerosis with leuco-erythroblastic anæmia, and peripheral pancytopenia with normally cellular or hypoplastic marrow but no marrow fibrosis. The groups are not sharply differentiated". The type of miliary tuberculosis in the four cases reported here was unusual in that histologically the lesions were those of miliary tuberculosis with little cellular reaction, but, in the three patients who died, they contained abundant tubercle bacilli. Similar cases have been reported in the years before treatment for tuberculosis was available. Paulley[2] treated by means of streptomycin and "Isoniazid" a girl

[1] *Quart. J. Med.* (1955), Vol. XXIV, No. 96, p. 351.
[2] *Brit. M. J.* (1954), 2: 511.

who had high fever and pancytopenia with no histological proof of the diagnosis, and apparent cure resulted, though the case at the time of reporting had been followed up for only a short time. In comparing the pancytopenic blood picture with other hæmatological abnormalities sometimes associated with disseminated tuberculosis, the authors postulate "that all these mechanisms may have a similar underlying mechanism, just as the corresponding primary disorders are now thought to be examples of a single 'myeloproliferative syndrome', perhaps due to a factor stimulating the bone marrow". They raise the practical question of the empirical treatment with streptomycin and "Isoniazid" of patients who have aplastic anæmia. In each of their patients fever was present; this is, however, a common feature of severe blood dyscrasias. There is no doubt that every case of pancytopenia—or for that matter every case of blood dyscrasia—should be investigated with determination and unfailing hopefulness. Only as a last resort should we fall back on that word of despair, "idiopathic"; to use it is to try to cloak our ignorance.

THE PAIN OF PEPTIC ULCERATION.

THE source of the pain in uncomplicated ulcer of the stomach or duodenum has been the grounds of considerable differences of opinion in recent years. A large ulcer may be painless and a small one may be excruciatingly painful. Pain disappears at the start of healing though the ulcer is still present. The relationship of pain to hunger and anxiety and the relief by antacid agents have led to views that pain is related either to local acidity or to muscular motility of the affected organ. Views are also held that pain is related to the local inflammatory reaction around the ulcer itself. Numerous papers have been written on the subject and many claims arising from physiological experiments have been made, but none of these have been satisfactorily substantiated.

A. W. M. Smith[1] has recently repeated both the studies on the effect of acidity and on the effect of motility upon the causation of peptic ulcer pain. Each of the 28 subjects investigated had typical epigastric pain relieved by food and alkali. By radiological means a duodenal ulcer was demonstrated in 16 of them and six others were found to have gastric lesser curvature ulceration. Gastric and duodenal motility was recorded by means of a kymograph attached to a pressure recording device; a balloon, manometer and Miller-Abbott tube were used. A Ryle's tube was sometimes fastened to the Miller-Abbott tube so that when the aspiration holes of the latter lay in the first or second parts of the duodenum, those of the former lay in the antrum or in the lower part of the body of the stomach. The apparatus was passed under radiological control into the stomach or duodenum and the balloon was inflated. Of the patients, 17 suffered from their usual pain when the balloon was inflated. The pain in most cases was not related to the presence or absence of local free acid as found by the aspiration of the gastric or duodenal contents at the time of inflation. In three cases there was good correlation between pain and acidity levels. In only two patients did pain appear to be sometimes associated with local motor activity and in the rest there was no correlation whatsoever. The effect of the administration by the intravenous route of antispasmodic drugs of the atropine group could in most cases not be correlated with the reduction in motility, for pain continued or ceased quite independently of the motor activity. Into the stomachs of 19 patients hydrochloric acid, 100 millilitres of a 0·5% solution, was slowly injected over five minutes. Fifteen patients developed peptic pain; in three this occurred after a second injection had been given. All the gastric ulcer and nine of the 13 duodenal ulcer patients developed pain and the average time before onset was 8·8 minutes. In each of five patients with duodenal involvement, injections into the duodenum rapidly produced pain. In only 12 of the patients did the introduction of acid result in an increase in motor activity, so that this secondary effect could not be the cause of pain. An injection in 18 patients of a 9% solution of sodium bicarbonate, 30 to 60 millilitres, invariably relieved the pain in an average time of nine minutes, though in half the cases the motility was greatly increased. The use of bicarbonate of soda instead of a more effective antacid will be questioned by some observers.

The summation of these results reveals possible inaccuracies in previous theories, though it adds little to positive knowledge. It is clear that motility is not a cause of the pain. Free acid is associated with the pain, but only in some patients, and pain may occur in the absence of any free acid. There was no support for the view that acid causes increased motility around the ulcer, nor was the relieving effect of sodium bicarbonate due to a decrease in motility. Every possible combination of local conditions was associated with pain or its absence. W. M. Smith suggests that the local effects result from changes in the sensitivity of the ulcer itself due to variations in the degree of œdema and engorgement of the ulcer area, themselves due, as Kinsella has suggested, to alterations in the blood flow. When the local inflammatory reaction results in increased sensitivity of the ulcer any mild stimulus will result in pain.

[1] *Quart. J. Med.*, October, 1955.

PLASMA PROTEIN CHANGES IN RHEUMATIC FEVER.

ONE of the difficulties in the treatment of acute rheumatic fever and in the detection of recurrences lies in the evaluation of the activity of the abnormal state. Alterations in the clinical signs are sometimes not sufficiently accurate to determine the delicate changes in response to therapy or to further streptococcal infection. While the clinical history and the stethoscope play the major part in the diagnosis and the ultimate prognosis of rheumatic fever, the subtle changes in the smouldering and flaring up of the whole process are so important, if the correct treatment is to be ordered, that a more critical method of detecting the rise and wane of the rheumatic tide is necessary. In this purpose, the determination of the sedimentation rate of the blood erythrocyte cells has served for some years as a fairly sensitive indicator of non-specific disease activity. The rate of accelerated sedimentation is not directly proportional to the degree of rheumatic activity, but varies from patient to patient. Thus, while in some patients the sedimentation rate increases to high values, falling finally only at the termination of activity, in others the rate rises only barely beyond normal values and falls to normal long before the rheumatic activity has ceased. The detection of recurrence is particularly difficult in patients with a resistant sedimentation rate so that undue optimism may arise. Conversely the sedimentation rate of erythrocytes is subject to so many influences that the lesser evil of undue pessimism may cause some confusion in treatment. Accordingly, attempts have been made to find a biochemical test more specifically related to rheumatic activity. G. H. Stollerman *et alii*[1] reported favourably on the use of levels of serum C-reactive protein as an indicator of rheumatic activity. The protein is identified by its capacity to form a precipitate with the somatic C-polysaccharide of the pneumococcus. C-reactive protein appears in the blood only in the presence of an inflammatory reaction. Serial determinations of C-reactive protein in patients with rheumatic fever revealed that the protein level was a very sensitive and reliable indicator of rheumatic activity if other pathological processes could be excluded. However, the C-reactive protein was not apparently elevated in the presence of chorea, *erythema marginatum*, subcutaneous nodules and myocardial Aschoff bodies. It is thus difficult to justify the assumption that the test would be any more useful than that of determina-

[1] *Am. J. M.*, November, 1953.

tions of erythrocyte sedimentation rate, especially if we consider the extreme simplicity of the latter procedure. Various suggestions have been made of the potential value of qualitative and quantitative estimations of plasma protein components. By the use of a cationic detergent technique for plasma protein fractionation it has become possible to make serial determinations of the various plasma proteins. R. F. Jacox[1] has used this determination in assessing the significance of plasma protein changes in acute rheumatic fever. Studies on healthy adults reveal that the amounts of albumin, fibrinogen, β-γ globulin and α globulin in the plasma vary little. By this method 60 patients with acute rheumatic fever have been studied. In each one, significant elevations of α globulin and of fibrinogen concentration were present in the acute phase of the disease, and at the same time the albumin concentration was depressed. The degree and persistence of the protein alterations usually reflected the severity of the initial attacks. In the case of four patients, comparisons between the erythrocyte sedimentation rate, the C-reactive protein titre, and the plasma protein changes were made. In each instance the α globulin concentration was the last to return to normal values. Subsequently in one patient the α globulin was increased on several occasions when the results of other tests were normal and the presence of rheumatic activity was confirmed by the electrocardiogram. Jacox concludes from these findings that evaluation of the α globulin concentration by cationic detergent fractionation is the most sensitive index of inflammatory activity in rheumatic fever.

How far the plasma protein changes may be regarded as specific indicators of rheumatic activity Jacox has not stated. However, there seems to be no reason for presuming that the plasma protein changes will not be subject to the confusing non-rheumatic fluctuations experienced by the C-reactive protein level and the erythrocyte sedimentation rate. While the newer tests may occasionally be of use in hospital practice, they would seem to add very little to the foundation of sound clinical judgement aided by the regular estimation of the erythrocyte sedimentation rate and the occasional use of the electrocardiograph.

ASSOCIATED GASTRIC AND DUODENAL ULCERS.

ALTHOUGH it is probably comparatively rare for radiological visualization of the stomach and duodenum to reveal the presence of concomitant gastric and duodenal ulcers, the finding is not so unusual in the post-mortem room. Widely differing figures have been quoted by surgeons and pathologists, so that the actual incidence of the occurrence is unknown. If it is agreed that the gastric and duodenal ulcers have a common ætiology, then the concomitant appearance of the two may be expected in a proportion of patients with the syndrome of peptic ulceration. However, arguments bearing on the common ætiology are vexed by considerable local differences in the conditions under which gastric and duodenal ulceration arise. For instance, the ulcerated stomach is frequently associated with low acid secretion and hypomobility and the ulcerated duodenum with a high acid level and hypertonicity. The importance of these factors in the causation of ulceration has recently been doubted, and it may well be that muscle spasm and changes in hydrogen ion concentration only irritate the already ulcerated mucosa, sensitized by local inflammatory reactions. Thus it would appear that although concomitant gastric and duodenal ulceration might be expected in the same patient, there are no obvious factors which will be likely either to initiate or to perpetuate both lesions at the same time.

H. D. Johnson[2] reports that of 347 patients operated upon for peptic ulcer, there were 32 with both gastric and duodenal lesions. Each of these patients was found to have the common factor of gastric retention. With the help of his colleagues, the author has investigated a further 103 cases of the concomitant ulceration. Most of the patients had a long history of dyspepsia. Of the cases, 30 were discovered on radiological examination and in seven neither ulcer was seen by such means. In the remainder, 21 patients had already been operated on for acute perforation of the duodenal ulcer before the gastric lesion was detected and most of them had been treated for the duodenal ulcer for several years. In all the cases, both on radiological evidence and on the findings at operation, it appeared that the duodenal ulcer was the initial lesion. All-night gastric secretion tests revealed that the general pattern in these patients was very similar to that of the typical duodenal ulcer type and that this included a high concentration of hydrogen ions in the gastric secretion. In 64% of the double lesion patients there was clinical, radiological or operative evidence of gastric retention. No evidence was obtained to confirm the view that gastric retention is followed by a fall in acidity due to secondary gastritis. The buffering of hydrogen ions which occurs in many cases of uncomplicated pyloric stenosis was not found in these patients. It is this inability to compensate for the retained acid secretion which may account for the gastric ulceration. The most frequent finding at operation was a healed stenosed duodenal lesion with an active, often huge, and sometimes bleeding, gastric ulcer. Other causes of the stenosis with secondary gastric ulceration included adult hypertrophic pyloric stenosis, anastomotic ulcers following gastro-enterostomy, and carcinoma of the pylorus. In five patients the secondary gastric ulceration occurred after vagotomy.

The secondary gastric ulcer developing as a complication of gastric retention is particularly prone to hæmorrhage, rapidly becomes large and deep and is particularly resistant to conservative treatment. The gastric ulceration may be multiple and may be the real cause of hæmatemesis in emergency surgical practice when duodenal ulcer is present. The author suggests that in the treatment of concomitant ulceration, operative procedure actually deals with an acid hypersecretion; the local resection of the gastric ulcer is insufficient. As an alternative to radical gastric resection, more limited resection may be combined with vagotomy, and with such a method there has been no recurrence. Operations likely to yield a small gastric stoma with secondary retention should be avoided.

[1] *New York State J. Med.*, March 1, 1956.
[2] *Am. J. Obst. & Gynec.*, March, 1956.

AIR POLLUTION.

SOME of the fogs which have been experienced in Sydney during the past few weeks are very similar to the London smog. As cities grow in size the probability of considerable air pollution increases. Philip Drinker[1] has discussed the problem of air pollution and its effects at some length. Some extreme cases of "smog" were seen in London in 1952, in the Meuse Valley area of Belgium in 1930 and in Donora, Pennsylvania, near Pittsburgh, in 1948. There were many deaths which could be put down to the air pollution in each case, and the general patterns of illnesses and deaths, including age distribution, were alike in all three.

In the Meuse Valley there were 63 deaths, in Donora 22, but in London where the "peasouper" fog lasted four days there were 4000 deaths. All age groups in London showed some increase in death rate, especially those over forty-five years. The commonest signs and symptoms were mucosal irritation, producing cough and sputum; bronchial obstruction, causing dyspnœa, cyanosis and loud rhonchi; cardiac failure in the elderly and constitutional upset. The deaths were mostly the result of bronchitis, pneumonia and diseases of the heart. The nature of the air pollution which brings about these results was fully investigated in London. During the 1952 smog sulphur dioxide gas rose from the normal (for London) 0·1 to 1·34 parts per million, and smoke (as weighable material caught on filter paper) from an expected 0·2 milligramme to 4·46 milligrammes per cubic metre. These substances alone cannot have been

[1] *New England J. Med.*, March, 1956.

responsible for the increased death rate. After the fog Londoners took a good while to clean up the greasy, black mess deposited inside and outside the houses. The evidence for the nature of the polluting substances in the Meuse Valley and Donora is indirect, for no observations were made at the time. In the smog-like fogs which occur in Los Angeles it was believed that sulphur dioxide was the cause, but this is a reducing substance, and a characteristic of the Los Angeles smog is its high oxidative power.

In London and many American cities air pollution is mainly due to burning of soft coal. In some places "scrubbing" of the smoke from soft coal fires has been carried out, but the evidence that this does any good is inconclusive. Much of the sulphur dioxide in smoke in America is now recovered as high-grade sulphur. Where there are smelters large quantities of substances as well as sulphur dioxide and sulphuric acid may pass into the air, the most dangerous being fluorine which is highly toxic to man, domestic animals and vegetation. Fluorine may also come from the production of phosphatic fertilizers from crude phosphate rock. A factor of some importance in the production of smog is the atmospheric phenomenon known as inversion. The air normally cools at about 5·5° F. for each 1000 feet rise. In inversion the air temperature gets a little warmer up to a maximum height of 300 to 3000 feet. This puts on a ceiling or lid whose boundary is clearly defined. During inversions, there is little or no wind and the plume from a smoky chimney comes out in a horizontal stream. Both the Meuse Valley and the Donora episodes involved several days of heavy natural fog, inversions and industrial pollutions. In Los Angeles smog is not infrequent. Here little coal is burnt, but very much oil and the little sulphur dioxide in the air help to reduce smog rather than increase it. Unburnt particles of petrol and products of its incomplete combustion undergo photochemical oxidation with the formation of ozone, which may occur to the extent of several parts per million. This reaction accounts, in a large measure, for the eye-irritating properties of Los Angeles air. Prevention of smog is not easy and each locality presents its own problems. In London the domestic open fire is the worst offender. In other places smelter pollution must be cut down, and this has been done very successfully in some places, notably Pittsburg. A trans-Atlantic aeroplane recently was unable to land anywhere near New York or Boston because of poor visibility, but was accommodated with acclaim by Pittsburgh. Sydney and other large Australian cities have had and are having warnings and should heed them. Something is being done, but apparently not enough. The biggest problem in all countries is the overgrowth of cities.

THYROID DISORDERS AND BREAST CANCER.

VARIOUS procedures have established themselves as forms of therapy additional to surgical removal of the tumour and its secondary deposits in cases of carcinoma of the breast. The relationship between the various hormones and the malignant cell is by no means clear, so that the newer treatments are based on empirical judgement backed by more or less strong statistical evidence of their success. The importance of the role of the thyroid gland in the development of breast cancer has been suspected for many years, though recent work has not involved this gland and attention has latterly been directed to the ovaries, the suprarenal glands and the pituitary gland. However, it has been suggested that cancer of the breast may be more prevalent in areas where goitre is common. While this does not necessarily imply a direct association between the thyroid activity and the malignant changes in the breast, it does raise the suspicion that there may be some obscure relationship. Certainly this could not be due either to the metabolic or to the carcinogenic action of thyroxin, as the incidence of carcinoma may be high where the output of thyroxin is low, and while in fact the phenomenon may be related to the compensatory action of pituitary hormones, the effects of thyroid hormone in breast cancer are worthy of study. A. G. Ellerker[1] has examined the subject from two points of view. On the one hand, a series of women who had previously undergone thyroidectomy were examined for evidence of breast abnormalities, and on the other, a series of women each with a histologically proven breast cancer were reviewed for possible history of thyroid disorder. In respect of the first series it was presumed that thyroidectomy resulted in a decrease of thyroid hormone. Of the 100 women who had previously undergone thyroidectomy, 32 had evidence of chronic mastitis, and in six, one breast had already been removed because of carcinoma. The incidence of breast cancer was thus 6%. Of this 100 women, 32 had irregular menstrual periods either before or after the thyroid operation, 15 had had an artificial menopause, and 33 had a family history of goitre, cancer, or uterine fibroids. Of the 157 women in the second series with breast carcinoma, 15 at the initial examination had been noted to have some enlargement of the thyroid gland. On review of the patients after recovery from the radical mastectomy, 12 were found to have evidence of thyroid disease, an incidence of 7·6%. In addition, three developed it during the follow-up period.

Several other statistical reviews have tended to show that abnormalities of the thyroid are significantly related to cancer of the breast. Ellerker suggests that a review of cancer in general gives the impression that hyperthyroidism and breast cancer are unusual together, and that the administration of thyroid extract in myxœdematous patients may result in regression of secondary deposits. The actual incidence of breast cancer in the general female population is unknown, but it is very unlikely to be as high as 6%, and the same reasoning presumably applies to the general incidence of thyroid disease in this reference, that of non-toxic goitre. If the inference is interpreted correctly, it would appear that after thyroidectomy patients should be maintained at the upper euthyroid level, and that in cases of breast cancer any tendency to hypothyroidism should be carefully controlled.

CORTISONE AND SERUM GAMMA GLOBULIN.

AMONG the several dangers attached to the use of cortisone is the apparent increased susceptibility of the patient to infection. Attention has been particularly paid to the possible flare-up of a quiescent tuberculous infection. However, that the phenomenon of increased susceptibility is not due to the action of cortisone on fibrous repair tissue has been shown by the clinical experience that the patient who is receiving cortisone may be susceptible to several infections other than tuberculosis. The mechanism of increased susceptibility is obscure, though it may be related to the relative leucopenia which has been found to occur in these patients, and which affects particularly the lymphocytes. R. S. Snell and T. Nicol[2] have investigated the effect of cortisone on the level of the serum globulin, with which are associated most of the antibodies to disease. The proportion of γ globulin in the total serum protein was assessed in 12 male guinea-pigs, each of which received a daily dose of 10 milligrammes of cortisone by the intramuscular route. The estimations of protein fraction were made at weekly intervals for five weeks. In each animal the level of serum γ globulin became reduced during the first two weeks of the cortisone injections and thereafter remained at low levels in all but two of the animals. It is to be noted that the dosage of cortisone was well beyond the therapeutic range, so that an exact parallel with human experience cannot be presumed. However, the experiments do show that cortisone may very well have an effect upon the level of serum γ globulin during treatment, and hence, that during such treatment the antibody defences of the patient may be profoundly disturbed.

[1] *M. Press*, April 4, 1956.
[2] *Nature*, March 24, 1956.

Abstracts from Medical Literature.

MEDICINE.

Treatment of Coronary Thrombosis and Myocardial Infarction.

J. E. WOOD, J. R. BECKWITH AND J. L. CAMP (*J.A.M.A.*, October 15, 1955) discuss the treatment of coronary thrombosis and myocardial infarction. They suggest that to evaluate therapy, some estimation of the severity or extent of the myocardial infarction must be kept in mind. Statistically the poor risk and the good risk patients can be separated. Two to three days after infarction, the poor risk patient may be found to have one or more of the following conditions: a history of previous infarction; intractable pain; severe and continuing shock; congestive heart failure; and an arrhythmia. The abnormality of cardiac rhythm may be auricular paroxysmal tachycardia, auricular fibrillation, auricular flutter, frequent ventricular ectopic beats from more than one focus, ventricular tachycardia, and heart block. In addition, the poor risk patient may have a history of the presence of thrombo-embolic phenomena, and a greatly enlarged heart. The good risk patient does not have any of these conditions. A reasonably large study indicates a mortality of 3·4% and a thrombo-embolism incidence of 1·3% in the good risk group as contrasted with a mortality of 60% and a thrombo-embolism incidence of 11·5% in the poor risk group. The authors consider that any argument for or against a given therapy must be aligned with some such evaluation of its usefulness. Pain must be relieved promptly; the best drug available for this is morphine. Atropine will not uncommonly relieve pain after a full dosage of morphine has apparently failed. Oxygen should be used for dyspnœa, cyanosis, shock, or when the control of pain is poor. The authors consider that no rigid control of diet is necessary, but in the first few days, liquids and a little toast to allay gaseous distension are probably all that should be given to ill patients. In good risk patients, and in poor risk patients who are improving, a low salt diet of simple foods ranging from 1000 to 1400 Calories is preferable to heavier diets. Generally, 2000 to 2500 cubic centimetres of fluid intake is sufficient. The bedside commode should replace the bedpan unless shock or debilitation of the patient prevents his using it. A statistical comparison of the results of chair and bed-rest methods of management shows that a large majority of patients with myocardial infarction can be treated effectively with the chair method. The patients are allowed to sit up, or are helped or lifted into the chair from the second to fifth day after infarction, but walking is not allowed. Indications are that there is less cardiac work done in the sitting than in the lying position. Shock complicating myocardial infarction has been correlated with a fatality rate of 80%, but the survival rate must be influenced to some extent by the nature and degree of the shock. A decision whether to use a pressor amine in a patient with borderline shock and with debatable blood pressure levels, may be reached by determining the amount of the urinary flow. Oliguria indicates the necessity for relief of the shock-like state. The advantage of arterial transfusion in myocardial shock has not been established. Depending upon the nature of the shock, therapy may frequently be carried out in the following order: the relief of pain by morphine and oxygen; the administration of atropine for bradycardia; the transfusion of blood or plasma for collapse of the veins; the administration of digitalis for congestion; the correction of arrhythmia with quinidine as in auricular fibrillation, paroxysmal tachycardia or ventricular tachycardia; the administration of neo-synephrine by the subcutaneous or intramuscular route as an early measure; and the giving of arterenol by an intravenous drip method for persisting shock. Intravenous drip therapy should be instituted within an hour after the onset of severe shock with oliguria. There is no unanimity of opinion as to which patients should receive anticoagulant therapy, though the authors consider that it should be given to every poor risk patient with myocardial infarction. However, the benefit to be derived from anticoagulants in the good risk cases is not great owing to the relatively low mortality and relatively low incidence of thrombo-embolic phenomena. When accurate laboratory facilities are lacking for the measurement of prothrombin time, the authors suggest that it is better to treat good risk patients by the bed and chair method than to subject them to the hazard of induced hæmorrhagic events. The choice of anticoagulant varies, but dicoumarol and phenylindandione are the most popular ones. The latter has the advantage of producing a therapeutic level of suitably reduced prothrombin time in one or two days, and the reduction is rapidly reversible in the event of undue prolongation of prothrombin time or hæmorrhage. A single injection of from five to 10 milligrammes per kilogram of body weight of vitamin *K* restores the prothrombin time to a near normal range in three hours. Generally, 100 milligrammes of phenylindandione given by mouth twice during the first twenty-four hours, followed by 50 to 75 milligrammes twice daily, will produce the effective lowering of the prothrombin level. Individual variation occurs and a smaller maintenance dosage is sometimes necessary. Anticoagulants are contra-indicated in patients prone to bleeding episodes, peptic ulcers, blood dyscrasias with hæmorrhagic tendencies and tumours of the gastro-intestinal or genito-urinary tract, and in patients with deficiencies of vitamin *K* or vitamin *C*. Wood *et alii* suggest that in acute coronary thrombosis with myocardial infarction, unless complicated by additional closure or extension, twenty-one to forty-two days should be a sufficient period for anticoagulant therapy, depending upon the individual patient. Digitalis should be given in most instances where there is a definite congestive failure with dyspnœa, increasing lung moisture, and filling of the neck veins. Administration of digitalis should be commenced early rather than late, though intoxication by this drug in these patients is hazardous and unwise. Control of arrhythmias is a matter of constant vigilance and prompt action, and the routine use of quinidine for some patients does not seem advisable owing to various unfavourable effects, including depression of myocardial function. In auricular flutter and fibrillation, digitalization should precede the use of quinidine to restore normal rhythm. Control of ventricular tachycardia by either quinidine or procaine amide is a matter of trial. The authors state that the prevention or control of paroxysmal or sustained arrhythmias in myocardial infarction is imperative.

ACTH and Cortisone in Severe Reiter's Syndrome.

D. T. FOXWORTHY, R. M. POSKE, E. M. BARTON, L. A. BAKER AND M. M MONTGOMERY (*Ann. Int. Med.*, January, 1956) discuss the use of ACTH and cortisone in the treatment of severe Reiter's syndrome. The condition is one of undetermined ætiology and is manifested by urethritis, conjunctivitis and arthritis. In its moderate and severe forms, it follows a characteristic course over a two to four months period, and no specific therapy is available. The antibiotics do not modify the course of the disease, which is self-limiting, although recurrences are not uncommon. Reports indicate differences of opinion on the value of ACTH and cortisone therapy in alleviating the urethral, conjunctival, joint and skin manifestations of the disorder. The authors have reported on the clinical findings and the results of treatment by hormones of ten patients admitted to hospital. It was found that although the course of the disease was not shortened by therapy, ACTH and cortisone, when given in adequate doses for a long enough period of time, were effective in suppressing symptoms. The intraarticular administration of hydrocortisone was of value in the few instances where it was used. No complications of systemic or local treatment were observed. While not curative, ACTH and cortisone are the most valuable agents now available to control symptoms in Reiter's syndrome, and should be used in severe cases.

Prognosis in Healed Subacute Bacterial Endocarditis.

C. E. MENDELSON, A. CAHUÉ, L. N. KATZ AND W. A. BRAMS (*J.A.M.A.*, February 11, 1956) discuss the prognosis for patients with healed subacute bacterial endocarditis. Twelve case histories are presented and a report is based on 17 patients in whom the diagnosis had been made on the clinical evidence and on positive blood cultures. In four patients the infection was superimposed upon a preexistent congenital lesion. The patients were observed for periods from six and a half to ten years after successful treatment. This disease, which was formerly almost always fatal, can now be healed by antibiotics in many cases; and permanent recovery can be expected in 65% to 75% of the treated patients. The histories of the present series show that some patients, especially if youthful, now recover without any residual disability. Seven patients have remained asymptomatic seven and a half to ten

years after recovery from infection. When aortic valvular regurgitation is the underlying disease, the prognosis seems less favourable. The patients were treated with penicillin.

The Psyche in Allergic Disease.

S. S. BURDEN (*Ann. Int. Med.*, December, 1955) discusses the role of the psyche in allergic disease. He suggests that the average allergist recognizes that emotional disturbances affect or even precipitate attacks of asthma or hay fever. However, there has been reluctance to delve deeper into the actual role that the psyche may play. As the treatment of allergic disorders by desensitization has been developed the role of the psyche has been forced into the background. The author agrees with Weiss and English, who have stated that "the allergic and the neurotic populations are so large that they must overlap". He also agrees that there is a relationship between neurotic character structure and allergic disorder. Case reports are presented of asthma, hay fever, urticaria and eczema, demonstrating the relationship of the psyche in these allergic disorders. It is contended that to be a good allergist it is necessary to be equipped with something more than just bottles of extract and syringes. The allergist should have the ability to blend allergic therapy and psychotherapy to meet the needs of the patient. When physical factors predominate, the allergist can produce the greatest benefit for his patient by applying sound principles of allergic investigation and therapy, as well as by establishing a doctor-patient relationship which will encourage the patient to speak of his emotional problems. When psychological factors either predominate or are the sole cause of the patient's illness, the physician must determine whether he is willing and sufficiently competent to treat the patient, or whether the patient should be referred to a psychiatrist. Most of the time it is necessary to continue allergic therapy while trying to break through the patient's emotional problem. Above all, the alert allergist should be able to distinguish the true allergic patient from the so-called para-allergic. If this is done, no patient need be subjected to unnecessary psychotherapy, or to prolonged and unnecessary allergic therapy. The conscientious physician will properly evaluate the physical and psychological factors in such a patient, and then treat him as he individually needs to be treated. Such an approach will result in much greater relief of symptoms for a great many people with allergic disorders.

Peptic Ulcer in Laennec's Cirrhosis.

W. P. SWISHER, L. A. BAKER AND H. D. BENNETT (*Am. J. Digest. Dis.*, October, 1955) discuss the high incidence of peptic ulcer in patients with Laennec's cirrhosis. In an investigation of 417 cases of Laennec's cirrhosis, peptic ulcer was found to be present in 13·9%; of these patients with peptic ulcer 28%, or 16, were admitted to hospital because of hæmorrhage from the ulcer. The study indicates that it cannot be assumed that hæmatemesis or melæna in a known case of Laennec's cirrhosis is always due to a ruptured œsophageal varix. The common finding of peptic ulcer associated with Laennec's cirrhosis makes it imperative that an accurate diagnosis be made as soon as possible in a patient with Laennec's cirrhosis, who is bleeding from the gastro-intestinal tract. It is probably a combination of factors which accounts for the increased incidence of peptic ulcer in these cases. Most important seem to be the direct stimulating and irritant actions of alcohol, the absence of neutralizing food, and perhaps a decrease in the flow of bile salts, and finally underlying psychiatric disturbances. The authors could find no evidence, in this series of cases, to incriminate portal hypertension, hypoproteinæmia, or hyperbilirubinæmia. The patient who is recovering from a prolonged alcoholic episode often has nausea, vomiting and abdominal discomfort. These may be attributed either to the direct and irritating action of alcohol on the gastro-intestinal tract or to the excessive preoccupation with self so often seen in alcoholics, and no further investigation of the gastro-intestinal tract is performed. Only when the ulcer is looked for is it found, and this may explain why there is so little evidence in the literature of this common finding.

Occult Enlargement of the Heart.

E. CORDAY *et alii* (*Dis. Chest*, November, 1955) point out that enlargement of the left ventricle of the heart is frequently missed on physical and radiological examination during life when that enlargement takes place in a downward direction. They show that if the patient drinks soda water immediately prior to X-ray examination, the large bubble of gas in the stomach thereby produced enables downward enlargement of the heart to be plainly seen.

Pathological Vagotonia in Emphysema.

O. A. ABBOTT *et alii* (*J. Thoracic Surg.*, November, 1955) have studied the effects of blocking agents of the vagus nerve upon the pulmonary vascular dynamics. One of their many findings was that the vagus nerve blocking drugs had the capacity to produce increased ventilatory capacity, decreased pulmonary arterial pressure and concomitant increase in oxygen uptake at rest and during stress in patients with pulmonary emphysema. From this it was deduced that a pathological degree of vagotonia is present in this disease.

Harmful Effects of Cough.

A. L. BANYAI AND M. JOANNIDES (*Dis. Chest*, January, 1956) emphasize the dangers of chronic, severe and uncontrolled cough. Destruction of a great many of the pulmonary alveoli and much of the elastic tissue is a usual sequel, especially in older patients. Emphysema commonly results and may be well established before it makes its presence felt.

The Heart in Cold Climates.

L. A. KUHN (*Am. Heart J.*, March, 1956) has investigated the effects of arctic climate and different shelter temperatures on the electrocardiograms of seven young men performing standard work out of doors. Significant electrocardiographic changes occurred with greater frequency and severity in the arctic than in a temperate climate, and the changes were more severe when the men lived in an arctic shelter at 50° F. than when they were housed at 70° F.

Acute Coronary Occlusion.

A. M. MASTER AND H. L. JAFFE (*Dis. Chest*, January, 1956) state their belief that anticoagulant drugs, administered during the premonitory phase of coronary thrombosis, are ineffective in that they neither prevent nor hasten the progress of the thrombosis. They believe that a low-calorie diet should be given in coronary thrombosis because such a diet diminishes the work of the heart and prevents gastrocardiac reflexes. Cortisone and corticotrophin are ineffective. Whisky should not be given, because it may increase the pulse rate. Smoking should be prohibited.

"Coin" Lesions of the Lung.

W. B. FORD *et alii* (*Am. Rev. Tuberc.*, January, 1956) have classified, according to their nature, the lesions present in the lungs of 124 patients who presented with well circumscribed tumours of the lung. In these cases the radio-opaque shadows were rounded like a coin and surrounded on all sides by lung. Of these lesions, 59 were benign (42 being non-specific granulomata), and 65 were malignant tumours. Of 729 other cases collected from the literature, 64% were benign (a large number being tuberculomata), and 36% were malignant tumours.

Prognosis in Laryngeal Paralysis.

E. G. HUPPLER *et alii* (*Am. Rev. Tuberc.*, January, 1956) state that in a study of 633 patients suffering from vocal-cord paralysis, no cause for the paralysis could be found in 181 cases. Of the remaining 452 patients, 273 had a post-thyroidectomy paralysis, 95 had a neoplasm, and 48 were suffering from cardiac or nervous disease. They state that when the cause of the paralysis is indeterminable and X-ray examination reveals no abnormality in the chest, the patient has a normal expectation of life and one chance in three of regaining his voice. When there are radiological abnormalities in the chest, the patient's chance of living five years is approximately three in five.

Need for a Long-Acting Tuberculin Test.

C. T. DOTTER (*Dis. Chest*, February, 1956) urges that a major investigative effort should be directed towards the development of a "depot" or long-acting test for sensitivity to tuberculin. With the best techniques now in use, the tuberculin test reveals only those individuals who have been infected more than four weeks prior to the time of testing. If an antigen could be placed in the body and retain its potency for several months or for a year or more, it would draw attention to the onset of infection at the earliest possible moment. The author is unable to put forward a method whereby such a test could be carried out, but suggests the introduction of the antigen into dental fillings, root canals or dental caps.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on March 20, 1956, the medical superintendent, DR. NORMAN H. ROSE, in the chair. The principal speaker was DR. J. M. YEATES, an honorary assistant surgeon of the hospital.

Clinical History.

The following clinical history was presented.

The patient was a married housewife, aged fifty-five years, whose history included the following symptoms: (i) Vomiting for five years. At first she had vomited about once each week, but for eight months prior to admission to hospital she had vomited at least once a day. This occurred at any time and was unrelated to meals. The vomitus was occasionally streaked with blood, and seven months earlier she had vomited a pint of dark-coloured blood. She had noticed black stools intermittently over the previous few months. (ii) Epigastric pain for eight months. This was sharp and stabbing in nature and passed through to her back. It was present "all the time", but was worse after meals and often woke her up at night. She vomited occasionally during the attack of pain, but this afforded no relief. Pain did not vary with posture. (iii) Constipation for "years". (iv) Breathlessness for three months when she was lying flat, but no extreme dyspnœa.

There was no relevant past or family history. She had attended the out-patient department two months prior to admission to hospital, and there a barium meal examination revealed "a fairly large hiatus hernia associated with fairly marked spondylitis in the lower thoracic region".

Physical examination of the patient revealed that she was a somewhat obese woman in no apparent distress. The only physical abnormality detected was tenderness in the epigastrium and in the right and left hypochondria. No masses were felt. There was no record of rectal examination. Other systems were normal. The blood pressure was 140 millimetres of mercury, systolic, and 100 millimetres, diastolic, and the urine was clear of albumin and sugar.

Examination of the blood showed 12·4 grammes of hæmoglobin per 100 millilitres, a white cell count of 6600 per cubic millimetre and a sedimentation rate of 12 millimetres in one hour (Westergren). Occult blood was present in the fæces. Chest X-ray examination showed a few calcified nodules in the left upper lung zone. No recent lesion was seen.

Medical treatment with antacids, antispasmodics, sitting up and washing down food with water after meals gave no relief. Œsophagoscopic examination showed a normal œsophagus with no reflux. Because of the persistence of pain it was decided to operate. Thoracotomy was performed under general anæsthesia along the eighth left rib, which was removed. A paraœsophageal hernia was found and returned into the abdomen. The right crus of the diaphragm was sutured to the left, and part of the diaphragm was sutured around the œsophagus. The lung was reinflated, and the wound was closed without drainage.

The immediate post-operative course was uneventful. The patient continued to complain of pain at the site of the incision. Six days later she commenced to have evening rises in temperature to 100° to 101° F., and this continued till she died.

Chest X-ray examination ten days after the operation showed fluid at the left lung base with localized pneumothorax above it and possibly some fluid at the right lung base. Pleural tap produced eight ounces of blood-stained fluid, from which was grown *Staphylococcus aureus* sensitive to all antibiotics except "Chloromycetin". Treatment with penicillin and streptomycin was started.

Pleural tapping was repeated twice. The patient's condition deteriorated, and seventeen days after operation the thoracotomy wound broke down, yielding a few ounces of pus. Air was sucking in through the wound, and the patient became severely shocked till a large dressing was held over the wound. Two hours later the wound was resutured, and drainage under water was instituted. Intravenous therapy with blood, glucose and saline was given post-operatively. X-ray examination later showed a partial pneumothorax and effusion on the left side and bronchopneumonia on the right side.

The wound broke down again, pressure drainage was applied, and further intravenous therapy was given. There was some reexpansion of the left lung apparent radiologically, but the patient died in peripheral circulatory collapse, four weeks after the initial operation.

Clinical Discussion.

DR. J. M. YEATES: The patient's history of vomiting is unusual in that it was so frequent and went on for so long. Five years is a long time, and there are many causes which could account for it. In general, we think immediately of some gastric lesion, and in a woman of fifty-five years we think first of peptic ulcer or gastric carcinoma. Early in her illness she had melæna, followed by hæmatemesis, and we can conclude from this that she had some form of ulcer. This would have been in the lower part of her œsophagus, in the stomach or the duodenum. It would be unusual for ulceration lower down to cause hæmatemesis. From the first paragraph of the history, therefore, we begin to feel that the patient had some sort of lesion between the lower part of the œsophagus and the duodenum; and whatever it was, it was almost certainly causing ulceration—anything from acute ulceration to carcinoma.

The pain was "stabbing". There is no mention of its being "burning". I feel that is important. The pain of peptic ulcer and also the pain of hiatus hernia, with which there is often œsophagitis, tends to be "burning". Mind you, often the patient's description is not very accurate. But "sharp and stabbing" is not exactly the type of pain that you get with peptic ulcer or acute œsophagitis. Passage of pain through to the back is not very helpful; lots of pains starting in the stomach and upper part of the abdomen tend to radiate to the back—for example, the pain of gall-stones, pancreatitis, any lesion in the stomach, even spondylitis, which you note she had. Her pain was constantly present, though worse after meals and sometimes at night. A duodenal ulcer often causes the patient to wake at night, rather more often than a gastric ulcer, and the same thing happens with gall-stones. As a matter of fact, the differential diagnosis between pain of gall-stones and that of ulcer is often almost impossible. Usually it is simple, but sometimes it is impossible even after all the tests, and only at operation can you make up your mind. From the point of view of the subsequent diagnosis of hiatus hernia we should comment on the fact that pain did not vary with posture. It is well known that in certain types of hernia the pain may increase when the patient stoops. This is due to reflux of gastric contents into the œsophagus, a thing which normally does not happen. The acid tends to burn the lower part of the œsophagus. On stooping or lying down, gravity assists the reflux, and pain is worse. We will see that she had a paraœsophageal type of hernia, and in that type as a rule they do not get much reflux. Thus we have two good reasons suggesting that her pain was not due to her hernia.

Long-standing constipation is common, and we can draw no conclusions from it. If it had been of recent onset, it might have been significant. Her breathlessness when lying flat fits in with the diagnosis of hiatus hernia. In this condition the stomach is liable to migrate into the thorax, usually the left side, so we get two sets of symptoms—those relating to the stomach and those related to the lungs, which, being pushed away, collapse. The reduction in vital capacity, accentuated when the patient lies down, may give rise to breathlessness. It is suggested that the hernia is a big one.

Perhaps we could see the barium studies now. You can see in the X-ray film a fairly large portion of the stomach filled with barium above the diaphragm. The films are not very clear. The spondylitis may have accounted for some of the pain.

Her obesity is significant. She had been vomiting for some years, and yet she is still obese. We can conclude that much of her food was still going through. In other words, she could not have had pyloric stenosis. We might think, too, that she could not have had a gastric carcinoma because she would have become thin. But this is a fallacious argument. As a rule patients with carcinoma lose their appetite—by the way, we are not told about her appetite—and they become thin, pale and pasty. However, we have all seen well-nourished persons who have been found to have carcinoma of the stomach. The least we can say is that if she had a lesion in addition to her hiatus hernia (always a possibility in this condition), then it was more likely to be non-malignant. Nor do we think she had pyloric stenosis. The only other physical abnormality was tenderness in the upper part of the abdomen. We cannot conclude much from this, as she was an obese woman.

Things we would think of are gall-bladder, stomach, pancreas and hiatus hernia.

The hæmoglobin level is reasonably good in spite of a hæmatemesis. It is worth remembering that patients with hiatus hernia can get anæmia without any obvious bleeding, and it should be borne in mind when we are confronted with a patient who presents with an obscure secondary anæmia. The white cell count is normal, and so is the sedimentation rate. The presence of occult blood in the fæces is of doubtful significance because of the questionable reliability of this test; if definite it would suggest that the lesion which previously caused hæmatemesis is still present. As for the chest X-ray film, so many people have calcified nodules in the lung areas that we can draw no conclusions from that finding.

The treatment, you will note, directed towards neutralizing the acid and having gravity assist the passage of food and acid, brought no relief. You might think that this is a point against peptic ulcer, but though treatment of this type usually helps them, it does not always do so. Some ulcers do not respond to even intense medical treatment. The negative findings at œsophagoscopy fit in with the para-œsophageal type of hernia, where the œsophagus comes down through the diaphragm in a normal manner and the sphincteric arrangements are really normal, but the cardiac part of the stomach goes up through the diaphragm as a hernia. It is just possible that a lesion at the lower end of the œsophagus, such as early ulceration, could have been missed. However, the chances are that she did not have any lesion there, which fits in with the absence of burning pain and the fact that posture, such as leaning forward, did not affect it. All this goes to suggest that she had no reflux of acid. One omission from the special investigations is a fractional test meal. This was not done, and I feel it should have been done. It is a very important test in peptic ulcer and also in hiatus hernia, and there are times when surgical treatment will depend on how much acid was found.

Anyway, as we see, the patient was operated on. I have illustrated on the board the techniques probably used in the operation. These herniæ have peritoneal sacs, and this would have been tied off as well. It surprises me, however, that no drainage was provided. I understand that the pleura is very apt to set up an effusion, even more so than the peritoneum, and I think I am right in saying that the accepted practice is to put in a drainage tube and connect it to an under-water bottle. It does little harm, is not difficult to instal, and you can then be sure that there is no accumulation of fluid. I thought that was practically routine. The continuance of pain is not unusual for a thoracotomy. The onset of fever six days after operation occurred at the usual time when troubles due to leakage arise.

Her later X-ray films show an increasing shadow at the left base, and no apparent shift of the mediastinum; the right side looks clear. They only got eight ounces of fluid, not a great deal, from which organisms were grown. This is an empyema, and it was treated with antibiotics, though we are not told whether any of this was put into the pleural cavity. I understand that the usual practice these days is to instil into the cavity some of the appropriate antibiotics. One case I had some years ago in this hospital following appendicitis responded very well to this treatment. After repeated tapping our present patient's wound broke down on the seventeenth day. This is later than the breakdown of abdominal wounds—you remember the old saying, bowel in the bed on the tenth day. There is little wonder that the patient was shocked, and the surgical repair and insertion of a drain must have been a most hazardous undertaking. Presumably it was done under local anæsthesia. The subsequent X-ray films show a partial pneumothorax, which is not surprising, some effusion and a good deal of trouble in the lung on the opposite side. Certainly there is not much vital capacity left, and it is not surprising that she was getting to be *in extremis*, becoming cyanotic and requiring oxygen. After suction drainage there was some reexpansion of the lung just before she died.

Let me sum up. On the clinical side, the story fits in very well with hiatus hernia. We might wonder about ulceration since she had a hæmatemesis, and it is practically certain that she had an ulcer. There is no evidence of portal hypertension with œsophageal varices. In hiatus hernia, ulceration is common. The infolded, angulated pouch of stomach is prone to superficial erosions going on to ulceration. As a result it is more likely than a normal stomach to bleed. Thus, everything up to the time of the operation can be accounted for. The next question is: are the post-operative complications sufficient to bring about her demise? I think most of us would agree that they are. The complications were severe and drastic. The lungs were almost without function—the left side collapsed and the right side with bronchopneumonia; and there was a pleural effusion with infection. Maybe, then, this was just a case of complications following thoracotomy. If this were so, how could they have been avoided, being wise after the event? First of all, I think, by not doing a thoracotomy. Thoracic surgeons tend to operate through the thorax and abdominal surgeons through the abdomen. That is natural, but there are many points in favour of an abdominal approach. The most obvious one is that when the abdomen is opened one can get a good look at the state of affairs. With a thoracotomy in the present case the surgeon had no idea what was in the abdomen. It could be that there was a mass in the stomach, that the patient had gall-stones, carcinoma of the stomach, pancreatitis, carcinoma of the pancreas, anything at all, as well as the hiatus hernia, which may not have been producing many of the symptoms. The greatest difficulty about hiatus hernia is to assess how much of the symptoms in any individual case is due to hernia. In that respect it is like a duodenal diverticulum. By the abdominal approach we can see what other lesions might be present and deal with the hiatus hernia as well. There are some herniæ, of course, particularly those on the right side, which are better dealt with from the thorax, but these are in the minority. Secondly, installation of drainage at the original operation may well have prevented the empyema.

I suggest that this patient had hiatus hernia and that she died from the complications, as described, of the operation. The other possibility is that there were extra complications which we have not heard about. I refer again to those under the diaphragm. The surgeon was dealing with a lesion which began below the diaphragm; so he must have been dissecting there, at some disadvantage as she was a fat woman, and this may have been difficult. She may have bled under the diaphragm from œsophageal or other veins. She may have developed infection there, perhaps a subphrenic abscess. There may even have been some ulceration in that area and the œsophagus torn by the manipulation.

The other possibility is that this patient had some additional pathological condition before the operation, as well as the hiatus hernia. There may have been a gastric ulcer or even gastric carcinoma; and although it may not have caused her death, it may have been an added burden. But there is no doubt that she had hiatus hernia and that the complications which followed an attempt to cure it were enough in themselves to bring about her death.

DR. ROSE: I think you could well ask why these harrowing and distressing complications of operation have been put before you today. These things are usually hidden away in the hospital archives. It makes me think there must be something very mysterious behind the story, and I should like to hear other people's impressions.

DR. W. MURPHY: I think Mr. Yeates has established that the patient died of complications following an operation for hiatus hernia. I, like most people, have been particularly struck by the association between hiatus hernia and other lesions such as duodenal ulcer and gall-stones. I suggest that one of these could have been present and could have caused her death, and I think we might find that there is something else in the stomach in this case.

DR. J. VOSS: There is only one point I wonder about, and that is the gas above the fluid in the pleural cavity. Whether that is air introduced during tapping, or whether it arose from a gas-forming organism, or whether it signifies communication between the airway and the cavity, we do not know. I incline to think that sutures might have passed through gut and pleural cavity. Quite frankly I think there is enough in the story as it stands to account for death, but if there is some obscure cause then I favour a tear in the lower end of the œsophagus or upper end of the stomach.

DR. W. EVANS: I was more interested in the original diagnosis which Mr. Yeates so ably discussed, rather than the later surgical complications. I wondered whether it was a straightforward case of hiatus hernia or whether, as Mr. Yeates said, there was some complicating factor. The points which seem relevant to me are that the woman had had blood-streaked vomiting and that she had had a severe hæmatemesis. I wondered whether one would have so much ulceration in a paraœsophageal hernia such as this. Ulceration occurs more often in direct hernia. There may have been some other site for the bleeding. The X-ray findings are negative, which is a point against, but does not exclude, ulceration or cancer. Her obesity and her vomiting make one wonder about her alcoholic history. There is no note of telangiectases or distended abdominal

veins, which one might expect if she had œsophageal varices, but it is a possibility.

DR. ROSE: What do you think of this case and the X-ray films, Dr. Calov?

DR. W. L. CALOV: One thing I have always felt is that if I had a hiatus hernia I would consult a physician rather than a surgeon and take some antacid tablets or powders. This has confirmed my feelings. There seems to be some collapse at the right base in some of the late X-ray films, but, of course, the films were taken with a portable machine.

When I first read the history I thought of carcinoma of the stomach, and there is a good deal to suggest this—dyspepsia, hæmatemesis, occult blood and so on. I thought of perforation, too, like Dr. Voss, but I think it would have been difficult to diagnose at the time. If it had been a perforation of the œsophagus, for example, there might have been surgical emphysema—though this may have occurred anyway. But I feel this is more than a good woman gone wrong as the result of thoracotomy. There is probably something other than the operation which has led to the complications, and I think carcinoma of the stomach is a possibility.

DR. ROSE: Dr. Palmer will give us the pathological findings.

Autopsy Report.

DR. A. A. PALMER showed slides and gave the observations made at post-mortem examination, of which the following is a summary.

The appearance of the body was that of an obese elderly woman. An unhealed thoracotomy scar twelve inches long extended along the line of the seventh left rib. An old lower mid-line abdominal operation scar was also present.

There was a left pyopneumothorax with thickening of parietal and visceral pleura. The left lung was partly collapsed and partly supported by adhesions.

The right pleural cavity was normal. A small abscess was present in the upper lobe of the right lung, and some branches of the pulmonary artery were filled with ante-mortem thrombus.

There was fibrinous pericarditis with a little thin purulent fluid. The myocardium was flabby, but the heart otherwise appeared normal.

The diaphragmatic hernia had been closed.

There was a perforated peptic ulcer on the anterior wall of the first part of the duodenum. The ulcer opened into a large localized peritoneal abscess.

A peptic ulcer was also present on the posterior wall of the stomach near the pylorus.

No other significant abnormalities were found. The uterus had been removed at a previous operation.

Microscopic Examination.

Right lung. Ante-mortem thrombus is present in large branches of the pulmonary artery and in some branches near the abscess suggesting that impaired circulation may have predisposed to the development of the abscess.

Stomach. The peptic ulcer is subacute.

Duodenum. As far as can be judged, this ulcer was also subacute.

Pathological Discussion.

DR. A. A. PALMER: As far as could be judged, the ulcers were of fairly recent origin, surrounded by young granulation tissue, probably not older than a couple of weeks. She died, remember, four weeks after operation. I do not think we can be sure from the histological appearances that the ulcers were present before the operation, and we have no clear indication in the history of the time when the ulcer perforated. Every now and then we find at post-mortem examination recently perforated peptic ulcers in patients who have been in hospital under treatment for other conditions which make it difficult to diagnose the rupture. It seems to be particularly lethal for an ulcer to perforate in hospital.

The association of peptic ulcers with hiatus hernia is not uncommon. I was interested to read that ulcers occur not only in the herniated part of the stomach, but they may occur anywhere.

Diagnosis.

1. Diaphragmatic hernia, treated by operation with subsequent left pyopneumothorax.
2. Ruptured duodenal ulcer with localized peritonitis.
3. Subacute gastric ulcer.
4. Right pulmonary embolism and abscess.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on October 5, 1955, at the Royal Melbourne Hospital. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital.

Phæochromocytoma.

DR. GEOFFREY A. PENINGTON presented a married woman, aged forty-eight years, who had been referred to the Royal Melbourne Hospital on September 1, 1955, for investigation. She had suffered attacks of palpitation for some fourteen years; they had been rather more severe for the past five years, and much more severe for the past six months. At first the attacks were of short duration, some lasting four to five minutes, beginning suddenly, and ending gradually. They tended to occur about four to five times a week, more often than not when the patient was working. About four to five years before she was referred for investigation the attacks had become more severe; they lasted longer, up to three hours, and were accompanied by tightness across the chest and upper parts of the arms, and by severe throbbing headaches, after which vomiting and/or retching occurred. Profuse sweating accompanied the defervescence of attacks. After such an attack she passed very little urine during the next twelve to twenty-four hours. The patient's relations were able to predict an attack, in that she first became quite white, her hands as well as her face; that was followed by flushing, palpitation, perhaps headache and vomiting, and then by a cold sweat. During the attack the limbs felt cold, and afterwards the patient felt worn out and stirred up.

For the past three years the patient had felt hot and sweaty between attacks, especially in hot weather. When those symptoms were very marked, they were associated with flushing of face and hands and a sensation of stiffness in the nose. Her appetite had been poor lately, and she had lost one stone in six months. Attacks occurred at any time, without rhyme or reason, on effort or at night. Her most severe attack was in May, 1955, and lasted three hours.

She had borne four children, the eldest being twenty-two years old and the youngest nine years. There had been no difficulties during the pregnancies, and no suggestion that she was hypertensive until June, 1955. Four years before admission to hospital she had undergone hysterectomy for a uterine polypus.

On examination the patient was observed to be thin, nervous and sweating. Her face was flushed, and her hands were pink and warm. Her blood pressure was 185 millimetres of mercury, systolic, and 110 millimetres, diastolic, on her admission to hospital. There was no cardiac enlargement, nor were any other abnormalities detected.

Her blood pressure tended to fluctuate between 230 millimetres of mercury, systolic, and 130 millimetres, diastolic, and 170 millimetres of mercury, systolic, and 110 millimetres, diastolic. On one occasion it rose sharply to 290 millimetres of mercury, systolic, and 140 millimetres, diastolic, this being associated with further retching and palpitation; it occurred during intravenous pyelography. The attack lasted for thirty minutes. She had intermittent glycosuria. Her basal metabolic rate was 14%. Her glucose tolerance curve was normal, with a urinary threshold of 130 milligrammes per 100 millilitres. Her blood pressure dropped from 220 millimetres of mercury, systolic and 125 millimetres, diastolic, to 170 millimetres of mercury, systolic, and 95 millimetres, diastolic, within one minute of intravenous administration of five milligrammes of "Regitine" (phentolamine). Excretion pyelography showed a rounded left suprarenal tumour, three and a half inches in diameter, compressing the renal pelvis. Urinary amines amounted to 300 microgrammes per litre (normal 20 to 40 microgrammes per litre).

The tumour was removed by Dr. Julian Smith on September 22, 1955. It was smooth, rounded, soft and fluctuant, about three and a half inches in diameter, and greyish in colour, with the suprarenal cortex stretched tightly across its surface. The operation and post-operative period were uneventful. Rises of pressure occurred upon induction of anæsthesia, and upon opening the abdomen; those were easily controlled with "Regitine". Intravenous injections of noradrenaline were administered post-operatively for the greater part of the first forty-eight hours in order to maintain a systolic pressure greater than 100 millimetres of mercury. Convalescence was smooth,

and the patient felt well, her systolic pressure varying from 120 to 140 millimetres of mercury.

Dr. Penington pointed out the diagnostic difficulty resulting from hysterectomy and the absence of indications that the menopause had actually occurred. The classical attacks were precipitated by pressure over the region of the tumour and were noted during the operation, at which times the administration of "Regitine" in the intravenous drip fluid was necessary. He said that in cases previously reported it had been noted that hypertension occurred particularly during the approach to the tumour, and its delivery or handling, and that hypotension promptly ensued when the venous return from the tumour was controlled by ligation of the veins. For the last-mentioned reason noradrenaline must be available for intravenous use at that stage. Continued administration of noradrenaline for about forty-eight hours was a common requirement. The fact that even minor attacks of flushing did not occur after the operation indicated that all the preceding symptoms were attributable to the tumour, which was innocent.

Carcinoma of the Parathyroid Gland.

Dr. Penington's second patient was a male industrial chemist, aged thirty-two years, who had been referred to the Royal Melbourne Hospital on December 5, 1954. During the previous twelve months he had lost more than one stone in weight; for six months he had found himself tired and irritable after work, and his energy was diminishing. Soon he had noticed headache, and his legs felt painful, especially with much walking or standing. For three months thirst and constipation had been troublesome, and epigastric pain unrelated to food, flatulence, pyrosis and occasional vomiting made their appearance. Frequency of micturition and nocturia developed, but there was no breathlessness or swelling of the ankles. Tiredness and weakness grew more oppressive until he was forced to his bed two weeks before admission to hospital. His previous health had been good, and there were no family illnesses of note.

On examination he was noted to be pale, and had evidently lost weight. His blood pressure was 135 millimetres of mercury, systolic, and 95 millimetres, diastolic, and his heart was normal. He had a small palpable spleen. His teeth, though not carious, were loose in their sockets, and his limbs were tender to the touch. A trace of albumin was found in his urine. His hæmoglobin value was 12·7 grammes *per centum*. His blood urea content was 70 milligrammes per 100 millilitres, and his serum calcium content 24·8 milligrammes per 100 millilitres. X-ray films showed generalized rarefaction of bone, with subperiosteal absorption of the phalanges, loss of the *lamina dura* and "ground glass" blurring of the skull. "Scout" X-ray films of the kidneys and barium meal X-ray examination showed normal findings.

He was allowed to go home for Christmas, but before he left hospital a soft tumour, about one and a half inches in diameter, appeared just behind the origin of the right sterno-mastoid muscle. After Christmas dinner he began to vomit—at first food, later coffee-ground material; this continued until his readmission to hospital on December 28, 1954, when he was extremely ill. His hæmoglobin value had fallen to five grammes *per centum*. He was given a transfusion slowly, and the lump was removed from his neck on December 31, 1954. Next day he seemed much better, and his improvement continued until he was allowed home two weeks later. This improvement was marked by rapid gain in weight, by falls in the serum calcium level from 18 milligrammes to 12 to 14 milligrammes per 100 millilitres and in blood urea level from 70 milligrammes to 33 milligrammes per 100 millilitres. The only symptoms experienced post-operatively were dyspepsia and tingling of the hands and feet.

His improvement was maintained after he left hospital, but was never complete. His weight remained around eight stone, his hæmoglobin value at 11 grammes *per centum* and his serum calcium level slightly elevated—12·6 milligrammes per 100 millilitres. The results of renal function tests were normal, though he had still some nocturia. He was still very weak and tired, his legs still ached, and he suffered severely from headache.

He was readmitted to hospital on April 12, 1955, complaining of severe and increasing weakness and of blurred vision. A soft tumour was felt deep to and behind the lower third of the right sterno-mastoid muscle. His hæmoglobin value was 12·7 grammes *per centum*, and his serum calcium content was 17·6 milligrammes per 100 millilitres.

The tumour was removed on April 21, 1955; it was pale, grey and fleshy, approximately one and a half inches in diameter, situated behind the inferior pole of the right lobe of the thyroid and adherent to this, to the right recurrent laryngeal nerve, to the prevertebral muscles, and to the œsophagus.

After operation the serum calcium content dropped rapidly to subnormal levels; the albumin phosphatase content was extremely high—about 80 King-Armstrong units. Tetany and paræsthesia were most distressing, and considerable difficulty was experienced in controlling them.

Calcium gluconate was given intravenously for about ten days, and then calcium lactate and dihydrotachysterol (AT10) were given by mouth together, and continued for almost two months.

His hæmoglobin value fell post-operatively to 9·7 grammes *per centum*, and this was corrected by blood transfusion.

He left hospital on May 28, 1955. He had then gained 10 pounds in weight, and his strength was returning. He was at first very depressed and unhappy, but he was able to return to work at the beginning of August, symptom free, his weight nine stone eight pounds and his bones recalcifying, with a normal serum calcium content and a persistent elevation of alkaline phosphatase content—30 units.

Dr. Penington pointed out the significance of bone and joint pains and fatigue as an indication of hyperparathyroidism and demonstrated the changes in radiological appearances associated with the condition and its control. Although there was no doubt that the tumour was carcinomatous, it was well differentiated.

At the time of the meeting the patient was working full time and feeling well, and there was no evidence of recurrence.

Torulosis.

Dr. Penington also demonstrated torulæ, which had been found by the clinical pathologist (Dr. Zimmerman) whilst examining the cerebro-spinal fluid from a man, aged twenty-eight years, who gave a history of headache and vomiting of three weeks' duration. The patient presented with the clinical manifestations of meningeal irritation and was submitted to routine investigations on the supposition that he was suffering from tuberculous meningitis. The second specimen of fluid obtained at lumbar puncture on the day after his admission to hospital contained the yeasts. They were also demonstrated in fluid obtained from puncture of the ventricles.

It was proposed to use dihydroxy-stilbamidine for treatment as soon as it was available.

A striking feature was a rise in pressure of the spinal fluid from 90 millimetres of fluid on the patient's admission to hospital to 200 millimetres after rehydration by means of intravenously administered fluids.

Mitral Stenosis Treated Surgically.

DR. L. E. ROTHSTADT showed a man, aged forty-five years, who had been admitted to hospital on July 22, 1955, following the sudden onset of severe headache and difficulty in speaking, and was rapidly becoming comatose. He was said to have had rheumatic fever about ten years previously, and for that condition he was in bed for a total of six months during the following three years. He had had shortness of breath on exertion over the year preceding his admission to hospital, and that had increased so that he had had to cease working as a chef. There had not been paroxysmal dyspnœa or hæmoptysis.

Examination of the patient on his admission to hospital revealed that he had mitral stenosis with auricular fibrillation, and also left hemiparesis. The latter was due to cerebral embolism. X-ray examination of the heart showed left auricular enlargement, but the left ventricle appeared normal in size. Electrocardiography confirmed the presence of auricular fibrillation, but the ventricular pattern was normal.

On August 31, 1955, mitral valvotomy was performed by Dr. John Hayward. There was no clot present in the atrium. The mitral orifice was one and a quarter centimetres long, and there was no calcification of the valve. A good split was obtained, the resulting orifice being three and a half centimetres long, and there was a slight regurgitant stream. After the operation auricular fibrillation persisted, but the murmurs had practically disappeared. The residual manifestation of the left hemiparesis was slight weakness of the muscles about the left angle of the mouth.

Dr. Rotstadt pointed out that the operation was advised because the man had the characteristic features of mitral stenosis with systemic embolism. The absence of clot in the atrium at operation was not unusual, as often it was recently formed clot that became swept away as an embolus, and older clot remained adherent to the walls of the chamber.

Post-Operative Hypothyroidism and Hypoparathyroidism.

The second patient shown by Dr. Rothstadt was a woman, aged fifty-five years, who had suffered from a large nodular goitre for some years. The goitre, which had been mildly toxic, had been treated by operation two years previously. The immediate course after the surgery was satisfactory until three weeks later, when she complained of tingling in the hands, feet and lips, with cramps in the calves. She was given calcium gluconate in doses of 30 grains, three times each day, and this therapy was continued for many months. In spite of this she had had paræsthesia, twitching of muscles and stiffness of her hands. She gained three stone in weight in the three months following operation, and with the administration of thyroid, half a grain daily, she lost one stone in weight. For a time she had attacks lasting two to three seconds, and in those she felt that her throat was constricted and she could not get her breath. She had been away from Melbourne for several months and without medical supervision during that period. In the last six months her eyesight had deteriorated, so that at the time of the meeting she could not read. Investigations on admission revealed the following findings: basal metabolic rate, −4%; serum cholesterol content, 160 milligrammes per 100 millilitres; serum calcium content, 7·2 milligrammes per 100 millilitres; serum phosphate content, 3·0 milligrammes per 100 millilitres; serum acid phosphatase content, four units; serum alkaline phosphatase content, eight units; total serum protein content, 7·0 grammes *per centum* (albumin 4·1, globulin 2·9 grammes *per centum*); hæmoglobin value, 106%; total leucocytes, 10,000 per cubic centimetre.

X-ray examination of the orbits, skull, left and right arms and urinary tract revealed no abnormal calcification or changes in bones. Renal function tests showed no abnormality. The blood urea content was 55 milligrammes per 100 millilitres, the percentage of urea being less than two in all specimens of small volume; the urea clearance (Fowweather) was 61%.

The ophthalmologist reported that there was an unusual type of opacity in the lens capsule of each eye, and he stated that he had seen one such case due to overdosage of vitamin *D*.

Since her admission to hospital she had taken each day one grain of thyroid and 120 grains of calcium gluconate. The dosage of AT10 had been gradually increased, so that at the time of demonstration she was having eight millilitres each day, and the serum calcium content was 8·8 milligrammes per 100 millilitres.

Dr. Rothstadt pointed out that the patient was suffering from post-operative hypothyroidism and hypoparathyroidism, and that she was responding to treatment. Operation on her eyes was contemplated by the ophthalmologist.

Acute Infective Polyneuritis.

Dr. Rothstadt then showed a man, thirty-five years of age, who worked as a gardener. He sprayed the garden with arsenical weed killer every three months, the last occasion being nearly four months before the demonstration. When he was admitted to hospital four weeks before the demonstration he had given a history of having had paræsthesia of the hands and feet three months previously; he had been treated at that time with vitamin tablets, and the paræsthesia had resolved in three weeks. He had remained free of symptoms until about four weeks before admission to hospital, when the numbness and tingling returned and gradually spread up to the groins and elbows. During the week prior to admission he had some difficulty in swallowing and phonation, together with paræsthesia about the lips and ataxia. His diet included very little meat. He had had poliomyelitis seventeen years previously. The muscles of the right shoulder had been mainly affected, with apparently full recovery after a few months. Examination of the patient at the time of admission to hospital revealed diminished power and diminution of tone in muscle groups of the upper and lower limbs. Tendon reflexes were absent, and plantar responses were flexor. Sense of position was absent in the toes, and there was some diminution of sensation to pin-prick and light touch in the limbs. The calves were not tender, but Rombergism was present. The cranial nerves were normal. Lumbar puncture revealed that the cerebro-spinal fluid was under a pressure of 130 millimetres of fluid. The protein content was 110 milligrammes per 100 millilitres, and there was a trace of globulin. The sugar and chloride contents were normal. No cells were seen.

Since his admission to hospital the patient had developed left wrist drop, with diminution of muscle power in the left hand. Examination of the hair for arsenic revealed four parts per million. Arsenic was not detected in the urine.

The diagnosis appeared to be acute infective polyneuritis, and the possible relation of the arsenic to the clinical features was discussed.

Aspiration Liver Biopsy.

DR. T. H. HURLEY showed two patients to illustrate two aspects of the diagnostic value of aspiration liver biopsy.

The first was a female patient, aged thirty-two years, in whom a diagnosis of miliary tuberculosis had been established in 1951 by aspiration liver biopsy. Treatment with streptomycin, isonicotinic acid hydrazide and para-aminosalicylic acid since then had resulted in a very satisfactory recovery. In February, 1950, the patient had become pregnant, and in August of the same year she had been observed to have ascites in association with her pregnancy. In November, 1950, she gave birth to a live normal infant, and after delivery was found to have considerable enlargement of both her liver and spleen. There was also gross ascites, and she began to run a high sustained fever. Two weeks later she was admitted to the Royal Melbourne Hospital. There her ascites was tapped and the ascitic fluid examined. On microscopic examination a few lymphocytes were found, and the protein content was shown to be 3·5 grammes *per centum*. Examination of her blood showed a hæmoglobin value of 65%, and a leucocyte count of 11,500 cells per cubic millimetre. A differential white cell count showed a marked "shift to the left" in the granular series. An X-ray examination of her chest showed widespread, moderately coarse mottling, suggestive of widespread bronchopneumonia. The result of a Mantoux test was negative with 1:1000 old tuberculin, but was positive when repeated with 1:100 old tuberculin. A liver biopsy was then performed, and examination of serial sections of the fragment disclosed the presence of typical miliary tubercles. The biopsy fragment was also incubated, and from it was grown *Mycobacterium tuberculosis*. In December, 1950, a course of streptomycin and para-aminosalicylic acid was commenced, and by April, 1951, her evening temperature had fallen to 100° F. In June, 1951, the course of therapy was discontinued, and in December of that year her liver and spleen were no longer enlarged clinically. In March, 1952, she again developed a fever, and a laparotomy was performed to drain a cystic collection associated with chronic tuberculous peritonitis. A further four-months course of therapy was given with streptomycin, para-aminosalicylic acid and isonicotinic acid hydrazide. In December, 1952, a further recurrence of fever and abdominal symptoms necessitated another laparotomy and drainage of an encysted fluid collection, and a further four-months course of therapy with streptomycin and isonicotinic acid hydrazide was given. Since then the patient had remained well and was now leading an active normal life. Her child had remained well from birth.

The second patient was a woman, aged fifty-two years, with sarcoidosis. The diagnosis had been established by liver biopsy. A left foot drop had occurred, probably as a complication of her sarcoidosis. The patient had first experienced dyspnœa with a tight wheezing sensation in her chest in January, 1954, and that had become progressively worse. In November, 1954, she had attended a mass chest survey unit, and the X-ray film had shown considerable mediastinal and hilar lymph gland enlargement. Clinical examination at that stage had confirmed the presence of a definite inspiratory and expiratory wheeze, audible at a distance from the patient, but no other abnormality was found. There were no palpable lymph nodes, and neither her liver nor her spleen was enlarged. Her Mantoux response was negative with old tuberculin 1:1000 dilution, and examination of her blood disclosed no abnormality. Her serum protein content and her albumin-globulin ratio were normal. Liver biopsy, performed by the transcostal route, showed the presence of multiple small granulomata scattered throughout the liver substance. Those consisted of endothelial cells, fibroblasts and collagen, with some peripheral collections of small round cells. No giant cells or acid-fast bacilli were seen, and there was no necrosis. The appearance strongly suggested sarcoidosis. In March, 1955, she suddenly developed a left foot-drop. Serial X-ray films of her chest from January, 1955, showed a progressive decrease in the size of her mediastinal and hilar lymph nodes, and in July, 1955, they had returned to normal size with considerable lessening of her dyspnœa. She had been given no specific therapy.

Gastric Biopsy.

Dr. Hurley's third patient was shown to demonstrate an aspect of the diagnostic value of gastric biopsy. The patient was a woman, aged fifty-five years, in whom subacute combined degeneration of the spinal cord had developed in the

absence of any detectable abnormality in her peripheral blood or bone marrow. The diagnosis was confirmed by a histamine test meal, a gastric biopsy and a therapeutic trial of vitamin B_{12}, which produced a satisfactory lessening in her symptoms. The patient had quite suddenly developed moderately severe dementia in 1952 after a fall one afternoon when she had been attending a race meeting. Investigations carried out at that stage had suggested that her dementia was arteriosclerotic in nature. In September, 1954, she noticed weakness, stiffness and numbness in both legs, and these changes were quite rapidly progressive. Six weeks later she was admitted to the Royal Melbourne Hospital. Examination at that stage showed her to be disorientated with regard to both place and time, with a very poor memory for both recent and remote events. There were the signs of spastic paresis, with early flexion deformity and bilateral extensor plantar responses. There was also detectable weakness in the grasp of her left hand and in her left biceps and triceps muscles. As far as could be ascertained sensation was reduced in both legs, but appeared relatively normal on the trunk. Investigations were undertaken with the following results. Lumbar puncture, X-ray examination of her spine, complete blood examination and sternal marrow biopsy all yielded completely normal findings. A histamine test meal revealed the presence of histamine-fast achlorhydria. A gastric biopsy, performed by Dr. Ian Wood, showed the presence of gross gastric atrophy compatible with pernicious anæmia. Between October 22 and November 9, 1954, while the investigations were proceeding, the patient's condition rapidly deteriorated. She developed the mental state characteristic of Korsakoff's psychosis, and she also became incontinent of both urine and fæces. On November 9 treatment with large doses of vitamin B_{12} was commenced. No increase in her reticulocyte count was observed, the reticulocytes being less than 1% of red blood cells on all occasions. The findings from sternal marrow biopsy performed on November 15, 1954, were indistinguishable from those before vitamin B_{12} therapy was commenced and were quite normal. On November 16, 1954, there appeared to be some improvement in the power of her legs, and on December 3 she was able to walk in calipers with assistance. By January 22, 1955, she had again become continent, and two weeks later she was able to walk without calipers and without assistance. Since then her condition had continued to improve, and she had become fully ambulatory and able to climb and descend stairs unaided. Tendon reflexes in her legs were still grossly increased, and both plantar responses were persistently extensor. Aside from some slight impairment in vibration sense in both legs below her knees, sensation was now normal. Intellectually she had remained facile with very limited insight, but her memory for recent and remote events had improved very considerably, and she was no longer disorientated or hallucinated. Treatment with vitamin B_{12} was being continued.

Temporal Arteritis.

The fourth case in Dr. Hurley's group was one of temporal arteritis in a man, aged fifty-seven years. He had been treated with cortisone. Details of the case were presented to illustrate the value of temporal artery biopsy in the early diagnosis of the condition, which was associated with disturbances of vision in about one-third of cases. Dr. Hurley pointed out that as temporal arteritis appeared in some instances to respond favourably to cortisone, early diagnosis and prompt treatment might be expected to reduce the incidence of the permanent and sometimes complete loss of vision which occasionally occurred. The patient had in November, 1954, noted the gradual onset of pain in the right side of his head. The pain gradually became more severe and extended to the opposite side of his head and radiated down the back of his ear and into his neck. Between October and December, 1954, he had lost 20 pounds in weight. He had noticed no visual disturbance. In December, 1954, he was referred to the Royal Melbourne Hospital as probably suffering from cervical spondylitis. On examination he was found to have readily palpable and tender temporal arteries on both sides of his scalp. Biopsy of the temporal artery of the left side showed the typical histological features of temporal arteritis. The wall of the artery was thickened, the lumen being partly occluded by a cellular type of granulation tissue. Areas of fibrinoid necrosis in the muscle coat were surrounded by large epithelial and giant cells. Administration of cortisone acetate, 75 milligrammes daily, was commenced. Some lessening in the pain in his scalp was evident within a week, and in three weeks it had disappeared and the temporal arteries were no longer tender. Cortisone acetate therapy was gradually discontinued after six weeks, and the patient had remained well since.

Breast Carcinoma.

DR. T. H. ACKLAND showed two patients who had been subjected to Urban's extended radical mastectomy for medial quadrant carcinoma of the breast. Portions of the second, third, fourth and fifth costal cartilages, together with a strip of the sternum, had been removed, and with these a length of the internal mammary vessels, in continuity with the main breast specimen. Since metastases to the internal mammary glands occurred in at least 60% of medial quadrant growths, it was suggested that the operation should be performed for carcinoma in that situation, when the patient's general condition was good.

Chronic Inflammatory Mastitis.

As a third patient Dr. Ackland showed a young woman, aged twenty-five years, suffering from non-specific chronic inflammatory mastitis. For several months a large hard mass had been present in the breast, and more recently softening, with subsequent abscess and sinus formation, had occurred. Biopsy showed an intense infiltration with inflammatory cells of many types. It was pointed out that the condition, which was unrelated to lobular hyperplasia, almost invariably resembled, in its early stages, an advanced carcinoma. Thus the desirability was emphasized of biopsy in all cases of breast tumour.

Gastrectomy.

Dr. Ackland also presented details of two cases of gastrectomy for bleeding duodenal ulcer, in which the duodenal stump had been deliberately drained by a rubber catheter. They were cases in which large posterior ulcers were present with no certainty that a safe closure of the duodenal stump would be possible. Drainage from the tube was at first profuse, but soon ceased in both cases. It was pointed out that the method might sometimes prove safer than accepting the risk of rupture, when the duodenal stump had been closed only with great difficulty.

Recurrent Lingual Thyroid.

Finally Dr. Ackland presented a female patient, aged fifty-three years, who had developed a recurrent lingual thyroid following removal, five years previously, of a similar tumour. On that occasion a portion of the nodule, which was then one and a half inches in diameter, had been deliberately left behind, the greater part being removed by splitting the tongue musculature from below. The recurrent nodule was approximately the same size when she returned complaining of dysphonia and dysphagia. It was completely enucleated by a lateral pharyngotomy approach. Investigation with radioactive iodine showed that no other thyroid tissue was present in the neck, and permanent thyroid therapy would be required. It was pointed out that a thyroid graft to a muscle bed, performed at the time of enucleation of the lingual thyroid, would have been a desirable procedure.

Ulcerative Colitis.

DR. E. S. R. HUGHES showed pathological specimens removed at operation from patients suffering from ulcerative colitis; he demonstrated the results of operative intervention in nine patients (a tenth was about to receive treatment); and a variety of ileostomy bags were made available for inspection.

Surgical Pathology.

Dr. Hughes said that knowledge of the disease had improved since primary colectomy and procto-colectomy specimens had become available for examination. Ulcerative colitis could exist in ulcerative and non-ulcerative phases; the right side of the colon was more resistant than the left in that condition; the presence of ulcers had serious pathological possibilities; the incidence of cancer in long-standing ulcerative colitis was high. From a study of 84 personal cases, the following classification was suggested: (i) proctitis, if the disease was confined to the rectum, and the sigmoidoscope could pass the upper limit; (ii) proctocolitis, if the upper level of inflammation could not be reached with the sigmoidoscope, whether the findings from barium clysma examination were normal or not; (iii) miscellaneous, if the lesion was atypical, such as segmental colitis and the so-called ileo-colitis.

Treatment.

Once macroscopic ulceration had developed, the need for surgery increased considerably. Medical measures would control the non-ulcerative phases of the disease. It was doubtful if psychiatry had any role in the management of the disease.

If surgery was required, permanent ileostomy and total removal of the large bowel were usually necessary. In most instances a colectomy could be performed at the time the ileostomy was constructed, and in selected cases, both the colon and the rectum could be removed as a primary procedure. Three patients demonstrated at the meeting had had an ileostomy and primary procto-colectomy.

In one case (the patient was a girl of sixteen years) the colon had been removed and the ileum anastomosed to the rectum. The patient now admitted to having seven or eight bowel actions daily, the stools being occasionally tinged with blood, but claimed that she was satisfied; sigmoidoscopic examination showed that the rectal mucosa had remained unchanged. The higher operative mortality and the risk of carcinoma in the rectal stump would deter many surgeons from attempting ileo-rectal anastomotic procedures.

Ileostomy.

Ileostomies with and without skin grafts were demonstrated. Dr. Hughes said that the incidence of ileostomy stenosis in the presence of a skin graft was depressing. Now that improved surgical conditions gave the surgeon more time, the surgeon could pay special attention to the construction of a neat ileostomy. The patients demonstrated at the meeting wore different types of ileostomy appliances according to their preference; one patient preferred a disposable bag, two others used "stick on" bags, whilst the others were content with simple rubber appliances.

Plastic Surgery.

DR. B. K. RANK showed four patients who had undergone operations for lymphœdema of the lower extremities. Two patients were used to illustrate the earlier Homan's type of staged operation, in which large flaps of tissue were raised and thinned by excision of the œdematous subcutaneous tissue and replaced. The other two patients illustrated more recent improvements on this technique. In them the whole stocking area of the skin and subcutaneous tissue from the knee to the sole of the foot had been excised and divided into three or four long rectangular strips; the skin had been removed from those strips with a Humby knife and then replaced immediately as free whole skin grafts. The procedure could be used only on patients who still had healthy skin. It had the advantage over the staged, more uncertain Homan's operation of being a single procedure with a dramatic and more certain immediate cure. Dr. Rank illustrated the difficulties of post-operative management he had encountered from "dermatitis" of the large anæsthetic area. That, however, seemed a transient phase.

DR. A. R. WAKEFIELD and DR. J. T. HUESTON showed a series of patients illustrating the repair of full thickness defects of the lip. A series of Abbé flaps from the lower lip was demonstrated for the correction of tight scarred lips resulting from the primary surgery of bilateral congenital lip clefts. It was emphasized that operation alone did not produce the complete correction of the deformity, and a special dental prosthesis was necessary to support the repaired lip. Defects of the lower lip exceeding one-third of its length, resulting from excision of carcinoma and radionecrotic ulcer, were repaired by means of a similar technique in which an Abbé flap from the naso-labial fold region was used. Patients were also present illustrating the rotation flap carrying the angle of the mouth into the lower lip with a later opening up of the angle, and the reconstruction of the whole lower lip by a visor flap from the neck by means of mucosal flaps from the cheek for lining and secondary buccal inlay graft.

Dermatology Demonstration.

DR. IAN STAHLE presented two patients. The first was a man, aged twenty-eight years. He had been a bricklayer for ten years and was suffering from chronic eczema of the hands. The first attack of eczema had occurred eighteen months previously, and he had not been completely free of the rash since then. However, he had continued in his occupation until five weeks previously, when the condition flared considerably after an unusually intricate job in which he had to chip old bricks most of the day in making a circular wall. He claimed that the intricate nature of the wall work was such that he was unable to take the usual care in avoiding contact with cement and that the brick dust had also irritated him. Two days before the clinical meeting patch tests were carried out with dilutions of potassium dichromate, an allergen frequently contained in cement. At the meeting the results of the patch tests were shown to be negative, and the skin of the fingers underneath the patch test areas were relatively free of the eruption. Dr. Stahle said that the relative freedom, after two days of covering, indicated that the chronic nature of the eruption was mainly determined by constant rubbing, and that even two days of occlusion were sufficient to produce considerable improvement. For several weeks the patient had used hydrocortisone ointment on one hand and a control base on the other. The hydrocortisone therapy was discontinued because the result was equivocal. Dr. Stahle discussed the difficulty of deciding when occupational aggravation ceased, and emphasized the fact that good results were rarely obtained unless all rubbing, including night and sleep rubbing, was eliminated.

Dr. Stahle's second patient was a man, aged thirty-five years. He had settled down in Australia three years previously and had worked in many different occupations. He was presented as a patient with a maladjusted personality who was always unhappy in his job and always looking for a better one. Fits of despondency would usually be associated with a flare in the constitutional type of eczema from which he suffered, and would occur irrespective of the nature of his occupation. As in the first case, Dr. Stahle said that the essential problem in management was to eliminate the harmful effects of night rubbing. In the present case his problem had been solved several times by the patient's admission to hospital, but once the skin had improved sufficiently for him to leave, further attacks of conscious or unconscious rubbing would occur, because he was psychologically unable or unwilling to face up to the problem of finding new accommodation and employment. Dr. Stahle said that the future of the patient depended on recognition of the fact that successful management and eventual rehabilitation depended mainly on sociological management with the aid of almoners and the full cooperation of his future employers and landlady. He said that unless the doctor maintained a vital interest in such patients, it usually happened that the almoner and other interested persons had insufficient control over environmental circumstances to continue to be of much use. He said that the difficulty of obtaining almoner services in such cases indicated the urgent need for expansion of those services.

DR. RALEIGH CLARKE demonstrated patients with *lupus erythematosus, adenoma sebaceum* and *granuloma annulare*. The patients were shown to indicate how easily a mistake in diagnosis could be made unless there were careful history-taking, full skin examination and awareness of the presence of the less common dermatoses.

The first patient, a woman, aged sixty-six years, had presented with fixed erythema over the dorsum of the fingers. The colour was influenced by room temperature, and the area was very itchy, particularly when warm or when irritated by primary irritants, such as soap and hot water. Casual consideration of the history and observation of the fingers would lead one to the diagnosis of eczematous dermatitis or possibly chilblains. Dr. Clarke demonstrated the absence of macules, papules and vesicles, which were characteristic of eczema. Two lesions on the face which to a casual glance looked like hyperkeratoses showed the same fixed erythema with mild scaling and minimal atrophy. The typical "carpet tacks" of *lupus erythematosus* were found to be present on lifting the scale. From the treatment viewpoint it was emphasized that, as in eczema, it was important to avoid contact with household irritants, as with any variety of dermatitis of the hands. Dr. Clarke said that superficial X-ray therapy was contraindicated and the use of chloroquine by mouth gave more satisfactory results than the older treatments such as intravenous injection of gold or intramuscular injection of bismuth. The patient suffered from mild hypochromic anæmia and arthralgia of the smaller joints. Those, together with the hand rash, demonstrated the connexion between fixed discoid *lupus erythematosus* of the face and the systematized variety of *lupus erythematosus*, the mortality of which had been reduced considerably with the use of modern steroid therapy. The patient was responding satisfactorily to chloroquine.

The second patient, a man, aged twenty-six years, had presented with wart-like lesions around several nail folds. There were also vertical ridges on the nails, and underlying those on the nail plate were similar wart-like lesions. The face of the patient showed the typical distribution of pink and yellowish papular lesions of *adenoma sebaceum*. Dr. Clarke said that that gave the clue to the correct diagnosis on the hands, namely, periungual fibromata, which were frequently seen in the condition. The patient was of normal mentality; but had he been subnormal or the doctor in a hurry, it was likely that the fact that the nail lesions had been present for many years in a static condition would have been overlooked, and the correct diagnosis would not

have been considered. Dr. Clarke said that the lesions in the present case were kept under control by caustic painting and diathermy and did not respond to X-ray therapy.

A patient with typical periungual virus warts was shown for comparison, to indicate the difficulty of differentiating several of the periungual fibromata from common warts on clinical grounds alone.

Dr. Clarke's last patient, a single woman, aged twenty years, had presented with a cluster of discrete pearly lesions over the knuckle; this cluster was 1·0 centimetre in diameter. Close examination revealed minimal atrophy and telangiectasia in the centre of the cluster. The margin was broken in three places, and the shiny, refractile, yellowish-pink to pearly, elevated edges suggested basal-cell carcinoma, *granuloma annulare*, dermatofibroma or xanthoma. There was also a simple dome-shaped, pearly pink nodule 0·75 centimetre in diameter on an adjacent knuckle. The lesions were painless and non-irritating. The lesions clinically were more typical of basal-cell carcinoma than of the other conditions, but the age of the patient and the atypical position called for a general skin examination, which revealed the typical annular and crescentic tumour formations of *granuloma annulare* on the dorsum of one foot. Dr. Clarke said that had the lesions presented on the neck and insufficient consideration or examination been given to the circumstances of the case, it was likely that the lesions, which were self-limiting, would have been removed by surgery or cancericidal dosage of superficial X-ray therapy. The chances of subsequent radiodermatitis and a severe immediate radiation reaction were greater than usual in this condition, which was histologically characterized by collagen necrosis. Dr. Clarke said that the lesson to be learned from the case was that doubtful skin tumours could be frequently diagnosed at a glance by a trained dermatologist and by most other doctors if they took the care to consider the rarer types of skin tumour which were benign, and which often could be diagnosed by full skin examination of the patient. It was not good practice and not necessary to perform biopsy of skin tumours on the face or exposed surfaces, such as the hands, unless every effort had been made to exclude the less common benign skin tumours which could occur at any age. Dr. Clarke said that the lesions of *granuloma annulare* usually retrogressed completely after several years, but individual lesions could be treated for cosmetic reasons and cured within two months by small doses of superficial X-ray therapy and/or the occlusion of the lesions with plain "Elastoplast" and light painting with a caustic such as trichloracetic acid.

DR. W. W. LEMPRIERE showed two patients with apparently similar eczematous lesions on their hands. Both patients were young married women. The first had a disorder of sweating, and the hands were irritated by wearing rubber gloves or by immersion in water or contact with soap, fruit juices or other household irritants. The second patient suffered from bronchiectasis and was in the habit of coughing over her hands. She was subject to repeated outbreaks of acute pyogenic infection on her hands, residual areas of apparent eczema being left. The practical problems of treatment in these two cases were contrasted.

Allergy Demonstration.

DR. R. H. O. DONALD, assisted by DR. D. BERMAN, DR. H. FRANCIS and DR. GWEN DONALD, presented a series of patients from the allergy clinic. Demonstrations were also given of skin-testing technique.

The first group of patients were suffering from hay fever and asthma. They had all undergone skin testing and been given appropriate desensitizing injections, with considerable benefit in most cases.

One patient suffering from hay fever and bronchial asthma had grown worse when treated with injections of house dust. However, the asthma had responded immediately to treatment with cortisone and had remained under control with maintenance doses of cortisone.

Another patient, who suffered from perennial hay fever and asthma, had not responded very well to desensitization injections. It was considered that in her case there was a very considerable psychogenic overlay. Excitement and worry would bring on attacks, and she fainted at the thought of being tested. It appeared that the psychogenic factor was of major importance.

A woman, aged sixty-one years, suffered from perennial hay fever bronchial asthma and migraine, as well as iodide-sensitivity. The results of skin testing were negative except for a slight reaction to house dust. Treatment with injections of mixed influenza vaccine B brought considerable relief from both asthma and nasal symptoms.

A man, aged forty years, had developed asthma four years previously. While in hospital he had been given penicillin and shortly afterwards developed a severe rash, which was still present. There was a strong family history of allergic disease. Apart from a slightly allergic appearance in the nose, the results of examination and skin testing were negative. His condition was regarded as one of penicillin sensitivity.

Two patients had been suffering from Ménière's syndrome. On being tested with histamine acid phosphate both had shown a strong reaction with pseudopodia. They had received benefit from treatment with "Hapamine" and histamine acid phosphate.

A woman, aged fifty-one years, had suffered from seasonal hay fever and recurrent attacks of migraine for many years. Skin testing had shown strong reactions to pollens, house dust and some animal danders, and a positive reaction to histamine acid phosphate. She was now being desensitized with histamine acid phosphate (1:1000), and her headaches had ceased in the last few weeks since she had reached a dosage of 0·7 millilitre and above. She would be desensitized with pollens later.

Malignant Neoplasms of Nose and Throat.

DR. GEORGE SWINBURNE, in conjunction with DR. KAYE SCOTT, showed a series of ten patients illustrating the treatment of malignant neoplastic lesions of the nasal cavity, accessory nasal sinuses, alveolar processes and hard palate by means of combined radiotherapy and electrosurgical exenteration. It was explained that a pre-operative dose of deep X-ray therapy approximating 6000*r* was given over six weeks. Between six and eight weeks later the surgical exenteration was carried out. The preliminary deep X-ray therapy dealt not only with the primary growth causing it to decrease in size, but also with the out-running cells in the actively growing periphery of the neoplasm. The resulting deformities were rectified by means of appropriate prostheses. The comment was made that one of the lessons learned in this type of work, which extended back to 1947, was that in ethmoid neoplasms it was better to sacrifice the eye and orbital contents on the side of the lesion and approach the neoplasm through the orbit. Not to do so was unnecessarily to endanger the patient's life further. Once that decision was made, the orbit concerned could be used to widen access by deep X-ray therapy also and the other one could be protected.

Dr. Swinburne also showed a series of eight patients who had undergone laryngectomy for extrinsic carcinoma of the larynx; in all cases the type of growth was epidermoid carcinoma. In two cases a block dissection of the lymph nodes of the neck had been carried out on the same side as the primary lesion, and at the time of the laryngectomy. The corresponding lobe of the thyroid gland was removed as well in those two cases. The series extended back to 1948. Particular interest lay in the patients' ability to use the pharyngeal voice, which had greatly improved since the laryngectomized patients in Melbourne had formed themselves into the "New Voice Association". That society was devoted to helping fellow members in all their problems and had made an enormous difference to the pre-operative and post-operative outlook of the people concerned. There was a regular roster of members who interviewed new patients pre-operatively. Another point of interest was the type of tracheal stoma and the evolution of the method of obtaining one when a permanent prosthesis (tracheotomy cannula) need not be worn.

Labyrinthectomy for Vertigo.

DR. RORY WILLIS presented the case histories of eight patients on whom labyrinthectomy had been performed by him for the relief of intractable vertigo of inner ear origin. He noted that in 85% of cases of aural vertigo the patients responded to conservative treatment, the remainder requiring some form of surgery. Surgery was not to be considered until the diagnosis was certain, and until a fair trial of conservative treatment had been given. There were five surgical procedures which could be undertaken for the relief of the condition—namely, (*a*) drainage of the *saccus endolymphaticus*, as advocated by Portmann in 1927, (*b*) intracranial section of the eighth cranial nerve, as recorded by Dandy in 1928, 1941 and 1951, (*c*) alcohol injection into the labyrinth via the ear drum and the foot-plate of the stapes (Wright, 1938), (*d*) labyrinthectomy, with use of a fenestration approach and technique well explained by Cawthorne in 1943, and (*e*) sympathectomy, as suggested and practised

by Garnett Passe in 1952. Only two of the procedures had stood the test of time—namely, intracranial section of the eighth nerve, and labyrinthectomy. The operation on the *saccus endolymphaticus* failed because the incision in the saccus to allow relief of intralabyrinthine tension or "hydrops" closed. The alcohol injection failed to destroy the labyrinth function in 50% of cases, and also carried quite a high risk of permanent facial nerve palsy. Sympathectomy failed to relieve the vertigo in 50% of cases.

In the comparison between the two acceptable operations, intracranial section of the eighth nerve was regarded as the operation of choice in all cases of bilateral labyrinthine vertigo, where "differential" section of only the vestibular fibres promised to preserve the hearing. It carried the usual risk of intracranial operations, *plus* a special risk of seventh nerve palsy in some 2% of cases.

Labyrinthectomy was considered the operation of choice in all unilateral cases of aural vertigo requiring surgery. It was technically safe, and required a short hospital stay. There was always total loss of hearing in the operated ear, but Dr. Willis noted that the residual hearing in patients with severe aural vertigo coming to surgery was so distorted that the value to the patient of retaining it was in most cases nil. He stressed that the operation should be performed by a "closed" technique, unlike the conventional fenestration; in other words, the approach should be by the posterior mastoid route, and the external auditory canal should not be opened. In that way the patient would not be left with a "mastoid cavity" requiring future attention.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

IMPURE MILK.[1]

[From the *New Medical Gazette*, July, 1875.]

THE progress of Scientific research has demonstrated that milk may become a medium for the propagation of enteric fever and other zymotic diseases and the analyses of samples of milk as a measure of precaution, has led in various instances to the detection of an adulterated and impure supply of that most important article of diet.

The question has recently been taken up by the authorities in Melbourne, and we have before us the report of the City Health Officer Mr. Girdlestone on the subject of the impurity of milk in that city. From examination of fifty samples, tonight in the City of Melbourne, Dr. Girdlestone considers nineteen to be genuine; seven poor and twenty four adulterated.

. . . . . .

The statistics of mortality in Sydney have been fearfully large in the last few years.

This may occasionally be accounted for by the unusually prevalent epidemics of zymotic diseases fed by the filth of Sydney, the imperfect drainage, and the contaminated water supply: but it still is brought home to us that the death rate of this city, abounding as it does in all the climatic influences ordinarily conducive to longevity, is in proportion to its population, fearfully high. The percentage of deaths of children as compared to births is astounding, and we often are pleasurably gratified by having the fact cast in our teeth by the numerous enemies who surround us, always on the alert for cause of complaint. To those of the medical profession who labour among the poor and middle class of the metropolis a grave cause of infantile mortality is revealed in the fact that the bodies of children are insufficiently nourished and their stamina powerless to withstand the brunt of the ordinary diseases of childhood. There are many hygienic causes adverse to the growth and general health of the youthful population of Sydney. The question of milk is evidently considered of too puerile a character to engage the attention of the Legislature at least in the Upper House. Mr. Charles Campbell having taken advantage of a motion for adjournment of the Legislative Council to put in a mild word in favour of an easy supply of good milk from Illawarra when the railway should be completed, several Honorable members most indignantly crushed him. Mr. de Salis who enjoys the reputation of philanthropy objected to the time of the House being taken up by remarks "on such a subject as milk". He did not consider "milk" a subject to adjourn the House upon. Mr. Docker thought the supply of milk from Illawarra would "prove a great boon provided the farmers did not drain the creeks" and the Hon Sir William Manning always gentle in his depreciation was "sorry to hear the railway to Illawarra mixed up with a question of milk". As the debate had arisen on a legal point regarding the payment of twelve shillings per week to an aged lunatic we may conclude that the legislators of our country consider the question of adequately nourishing our large infantile population of vastly less paramount importance than the support of one unfortunate imbecile. There appears to have been a "wondrous kindness" in the "fellow feeling" for the poor old lady, contrasting strangely with the apathy regarding the supply of a life-giving nutriment for the support and health of the rising generation.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

HYPOTENSION AFTER "THIOMERIN" IN A PATIENT RECEIVING "SERPASIL" AND "ANSOLYSEN".

SIR: This is to report a great and prolonged fall in blood pressure, occurring in a patient already taking "Serpasil" and "Ansolysen", each injection of "Thiomerin", one cubic centimetre, causing a fall of about 120 millimetres of mercury, systolic, and 50 millimetres, diastolic.

A housewife of fifty-four had originally a blood pressure of 280/130. With "Serpasil", 0·25 milligramme *t.d.s.*, it fell to about 225/100 (range 225-255/95-110).

With "Ansolysen", 40 milligrammes *q.d.s.*, and "Serpasil", 0·25 *q.d.s.*, the blood pressure was about 215/90. Twenty-four hours after 0·5 cubic centimetre of "Thiomerin", given by intramuscular injection, the blood pressure was 175/65. Twenty-four hours after one cubic centimetre of "Thiomerin" the blood pressure was 115/55 sitting and 125/65 standing. Mild hypotensive symptoms occurred about twelve hours after the injection, when the pressure would presumably have been less than 100/50, and four days later the blood pressure was still only 155/70. This effect has been confirmed in this patient on seven occasions.

In effect, hypotensive symptoms begin about ten hours after injection and persist until about thirty hours after. The maximum fall is about twenty-two hours after the injection, and the effect is passing off on the fourth or fifth day.

It would be interesting to know if any others have observed this effect.

Since the initial observation this effect has been demonstrated in four other patients. The average duration of hypotension is three days after 0·5 cubic centimetre of "Thiomerin" and four to five days after one or two cubic centimetres.

The effect is also observed after "Diamox", though it does not appear to be so reliable.

Hypotension occurred after "Thiomerin" in one patient on "Serpasil" without "Ansolysen", but it did not occur in a patient given "Thiomerin" alone.

Yours, etc.,

A. MCQUEEN THOMSON.

100 Collins Street,
Melbourne,
June 21, 1956.

THE IATROGENIC STATE.

SIR: I would like to draw the attention of the members of our profession to a common occurrence which is quite well known, but of which we need to be reminded.

I have in mind iatrogenia. Iatrogenia is a condition in which a patient develops a number of symptoms due to faulty handling by a doctor.

We have to admit that quite often we consciously or unconsciously say a word or make a gesture which will not

soothe an anxious patient, but rather develop deeper and further anxiety about his health. Sometimes the patient receives treatment in such a way that dangerous habits are formed—morphine addiction is an example.

Those states of iatrogenia which one could classify in the group of anxiety neurosis or anxiety states are predominant when a patient has the misfortune to suffer from a condition which is quite obscure medically or which is not fully understood.

As a neuropsychiatrist I think that a lot of harm can be done and probably is done, especially with patients who have compensation claims, when the compensation presents a major psychological disturbance, and it is the expectation of money which causes anxiety—as in compensation neurosis. This harm can be largely avoided by care in every word and gesture in the presence of a patient. A specialist of any branch of medicine or a general practitioner is always a psychotherapist whether he is aware of it or not.

Yours, etc.,

OSCAR R. SCHMALZBACH.

"Harley",
143 Macquarie Street,
Sydney.
June 21, 1956.

SPIDER BITES IN AUSTRALIA: AN INVESTIGATION.

SIR: It will be appreciated if you will bring to the notice of your readers that in connexion with investigations which are being conducted at the Commonwealth Serum Laboratories on the toxicity and antigenicity of venomous spiders, we desire to collect data on the incidence and clinical effects of spider bites in Australia.

We invite medical practitioners who have treated cases of spider bites in recent years to communicate their experiences to us.

Yours, etc.,

F. G. MORGAN,
Director.

Commonwealth Serum Laboratories,
Parkville, N.2,
Victoria.
June 18, 1956.

A MEMORIAL TO THE LATE W. A. HAILES.

SIR: A number of the friends and admirers of the late W. A. Hailes feel that his name should be perpetuated in some tangible way at the Royal Australasian College of Surgeons. He was an ardent worker for the College, closely associated with its early history, and intensely interested in its future. It is proposed to present some dignified article of furniture, suitably inscribed, to the College as a mark of our esteem for his life of hard work, friendly loyalty and devotion as a teacher, surgeon and army consultant.

I have received a number of cheques for this purpose from his many friends over a wide circle of hospital associates, past residents, and men who had served under his direction in the army. As such people are now scattered far and wide over the Commonwealth, one is not able to contact them all personally; but if you could bring this matter to general notice through your columns it would enable any of those who would like to associate themselves with this gesture to do so. If they forward their cheques to me I will be pleased to add them to the total.

Yours, etc.,

B. K. RANK.

59 Collins Street,
Melbourne,
June 18, 1956.

A SPECIAL UNIT FOR CANCER RESEARCH IN NEW SOUTH WALES.

SIR: All those responsible for the establishment of the Special Unit for Investigation and Treatment are very grateful to you for drawing attention to the unusual and worthy character of the undertaking.

It was the wish of the New South Wales Government that the project should be carried out "with the utmost expedition". That this has been achieved has been largely due to the remarkable enthusiasm of the Medical Superintendent of the Prince of Wales Hospital (Dr. R. F. Kaye-Webster) and of the building contractor (Mr. A. R. Hinwood).

The project will be stillborn unless it receives at once from medical practitioners those cases suitable for study. At the commencement black moles, lumps in the breast, suspected carcinomata of the stomach, colon and rectum and enlarged lymph glands due to reticulosis are being sought for investigation and treatment as from Monday, July 23, 1956.

It is to be hoped that as the result of the prominence given to this new venture in your leading article a ready response will be forthcoming.

Yours, etc.,

KENNETH W. STARR,
Honorary Director.

149 Macquarie Street,
Sydney,
June 22, 1956.

THE WONDER DRUGS OF PSYCHIATRY.

SIR: May I wonder in print about ataraxics, out of drug house Greek by the *Zeitgeist*?

Referring to Dr. Morgan's monograph "The Use of Reserpine, Chlorpromazine and Allied Drugs in Medicine and Psychiatry" (M. J. AUSTRALIA, June 23, 1956), it should be noted that daily doses of the wonder drugs and their adjuvants would cost the patient (or, if regulations are amended, the taxpayer) about £250,000 for the forty or fifty years the usual asylum "lifer" would need them. (Check my arithmetic, sir, with any drug house, and the doses with the monograph.)

If this quarter of a million pounds is compared with the mere £30,000 that Dr. Cunningham Dax estimates it costs for mental hospitalization of a similar psychotic (nursing and administration thrown in), I still agree with the physician in "Macbeth" (Act V) in both his major conclusions.

However, on the centenary of the birth of Sigmund Freud, the date of the publication of this article, one reflects that there are very few cells indeed "above the superior *corpora quadrigemina* in the mesencephalon". Although Freud, too, let his pen run away with him, he started with a detailed knowledge of the anatomy of the central nervous system. I think he knew too much about histology to put his "id" and "super-ego" in the *formatio reticularis* of the mesencephalon; a particularly calamitous error after Descartes's *faux pas* with the *corpus pineale*, a closely adjacent structure, and equally inaccessible to the ataraxics functionally.

Yours, etc.,

GODFREY HARRIS.

607 New South Head Road,
Rose Bay,
June 28, 1956.

THE MEDICAL BENEFITS FUND OF AUSTRALIA AND SERVICES RENDERED IN PUBLIC WARDS OF HOSPITALS.

SIR: It has come to the notice of the Executive Committee in New South Wales of the Medical Benefits Fund of Australia that there is a disposition in some members of the profession, especially in country districts in this State, to hold the Fund responsible for the failure to pay benefits to contributors for services rendered in public wards of hospitals. It has been reported that statements have been made in local association meetings that the State Government is willing that charges might be made for these services to contributors, but that the stumbling block is the Medical Benefits Fund. With the concurrence of members of the Executive Committee I desire to state that such statements are completely incorrect.

The relevant facts are as follows:

1. The Commonwealth Government will not permit benefits to be paid for services rendered in public wards, and in any case will not permit benefit to exceed 90% of the fee. The Government has instructed the Funds accordingly and has required the Funds on their claim forms to request the contributor to state whether the patient was classified as

public, intermediate or private, so that this instruction may be proved to have been observed in settlement of claims.

2. The State Government has informed the British Medical Association that it will not agree to public ward patients being charged even the difference between the benefit and the fee.

3. The Medical Benefits Fund, although willing to settle on the basis of the 90% rule to contributors classified as public, is prevented from doing so by these decisions of Commonwealth and State Governments.

4. The 90% rule has been approved by all competent authorities, including the British Medical Association.

5. In its administration the Medical Benefits Fund is strictly bound by the terms of the *National Health Act* in this and in all other related matters.

The Council and the Executive Committee of the Fund have at all times and in all circumstances endeavoured to make their decisions conform, so far as the *National Health Act* will allow, to the just interests of contributors and doctors rendering the services.

H. R. R. GRIEVE,
Chairman.

Caberfeidh,
113 Homer Street,
Undercliffe,
New South Wales.
July 2, 1956.

Obituary.

JACK MORLET.

WE have received from Dr. C. R. R. Huxtable the following tribute to the late Dr. Jack Morlet.

Dr. Jack Morlet, whose death was reported from Ringwood, Victoria, in April, was a veteran of the two World Wars. As an old friend, and as a fellow member of "Kitchener's Hundred", as we used to like to call ourselves, I should like to pay my tribute to his memory.

In early 1915, Lord Kitchener was preparing his new army for the four years' struggle which he, along with very few others, was bold enough at that early stage to prophesy. He appealed through the War Office to Australia for a hundred medical men for work with field ambulances, and with regiments. Most young men in those days considered it something of a privilege to be chosen for such work. In the early stages of the second World War with all its disillusionment, many young medical men considered such volunteering to be sheer stupidity. Who shall say which attitude is the better? To my mind one thing is certain in the matter—the group in which one will find the more lovable comrades. Jack Morlet was such a one. Enthusiastic, humorous and kindly, he was a general favourite with the forty men from Victoria. In camp together at Eastbourne, the summer beauty of the Sussex Downs made us forget the uncertainties ahead, and in retrospect seemed an idyll of delight. From there he was sent to serve as regimental medical officer to the Second Battalion of Northumberland Fusiliers of the 27th British Division in Macedonia, where he spent the next few years in that fever-torn country, the most disease-stricken of all the war zones.

Dr. W. K. McIntyre, of Launceston, in his recent interesting address "Reminiscences of the Royal Army Medical Corps in Macedonia in the First World War", describes the ravages of malaria (just as I have heard Jack Morlet describe them in days gone by)—an army of "listless, anæmic and sallow men, whose lives were a physical burden to them". How wonderful it was that those same armies were able to attack and to achieve a complete decisive victory in September of 1918, six weeks before the German collapse on the Western Front!

Jack Morlet had a great affection for the British soldier and for the unit with which he served.

Awaiting demobilization in England, he took a house surgeon appointment for six months in a London hospital. Most people were war-weary in those days and impatient. One particular honorary surgeon, whose war work had been done in comfort on the home front, made the mistake one day in the operating theatre of speaking in a bullying and offensive manner to Morlet, his assistant. Perhaps a recent graduate might have been cowed and might have taken it—not so Jack Morlet. The answer he gave caused a chilly silence to descend upon the operating theatre, and at the inevitable interview afterwards Jack Morlet's gentlemanly attitude caused the other man to realize his mistake and to apologize.

Back in Victoria, Jack Morlet practised for many years at Camperdown. In the second World War the old spirit flared up, and he was soon away overseas on sea-transport work, in which he had some lucky escapes, as his ship left Malaya only a few weeks before the fall, and on a later occasion he was in Darwin harbour during the big Japanese air raid. In later years he had practised quietly at Ringwood in Victoria, having suffered in the interim several major operations and much ill health.

He leaves a widow, a son and a daughter. Amongst his old friends he leaves a memory of loyalty and of warmth, of songs and of laughter, and (until his later years of ill health) an enthusiasm for living that was fresh and youthful.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 33, of June 21, 1956.

NAVAL FORCES OF THE COMMONWEALTH.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointment.—Charles Joseph Avison Parker is appointed Surgeon Lieutenant, dated 21st February, 1956.

Promotion.—Surgeon Lieutenant Frederick Arthur Stenning is promoted to the rank of Surgeon Lieutenant-Commander, dated 4th May, 1956.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—The age for retirement of 1/43713 Lieutenant-Colonel A. Fryberg, M.B.E., is extended until 26th May, 1957.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The provisional appointments of the following officers are terminated: Captains F3/1019 P. B. Blackall, 24th December, 1955, and 3/149015 M. C. Piercy, 10th January, 1956. To be Captains (provisionally): F3/1019 Pamela Beatrice Blackall, 25th December, 1955, and 3/149015 Murray Chandler Piercy, 11th January, 1956.

Central Command.

Royal Australian Army Medical Corps (Medical).—The age for retirement of 4/35230 Lieutenant-Colonel R. T. Binns, O.B.E., E.D., is extended until 12th June, 1958. 4/31955 Major H. R. Moore is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical) (Central Command), 19th April, 1956.

Western Command.

Royal Australian Army Medical Corps (Medical).—5/26543 Captain J. M. McKenna is appointed from the Reserve of Officers, 26th March, 1956.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

Northern Command.—To be Honorary Captains: Ronald Neil Brown, 20th April, 1956, and Terence Higgins Brumm and Margaret Ann Hickey, 23rd April, 1956.

Eastern Command.—Honorary Captain E. H. Miles is retired, 16th April, 1956.

Southern Command.—The age for retirement of Major D. E. Gowenlock is extended until 20th June, 1957. Honorary Captain M. J. Blankenberg (*née* McCulloch) is retired, 7th May, 1956.

Central Command.—To be Honorary Captain, 24th April, 1956: Alan Bentley.

Medical Practice.

NATIONAL HEALTH ACT.

The following notices appeared in the *Commonwealth of Australia Gazette*, Number 33, of June 21, 1956.

NATIONAL HEALTH ACT, 1953-1955.

Part IV: Pensioner Medical Service.

Reprimand of Medical Practitioner.

I, Donald Alastair Cameron, the Minister of State for Health, hereby give notice, in pursuance of subsection (1.) of section 134A of the *National Health Act* 1953-1955 that I have this day reprimanded Noel Lyster Hall, of 78 Racecourse-road, Ascot, medical practitioner, following investigation and report by the Medical Services Committee of Inquiry for the State of Queensland established under section 110 of the *National Health Act* 1953 concerning the conduct of the aforesaid medical practitioner in relation to his provision of medical services for pensioners and their dependants under Part IV. of the *National Health Act* 1953-1955 or under an arrangement made by the Director-General of Health under section 7 of the *National Health Service Act* 1948-1949 and the National Health (Medical Services to Pensioners) Regulations.

Dated this 31st day of May, 1956.

DONALD A. CAMERON,
Minister of State for Health.

Part VII: Pharmaceutical Benefits.

Reprimand of Medical Practitioner.

I, Donald Alastair Cameron, the Minister of State for Health, in pursuance of the powers conferred on me by subsection (1.) of section 134A of the *National Health Act* 1953-1955 hereby give notice that I have this day reprimanded Francis Swiggs, of Cleve, medical practitioner, following investigation and report by the Medical Services Committee of Inquiry for the State of South Australia.

Dated this 31st day of May, 1956.

DONALD A. CAMERON,
Minister of State for Health.

I, Donald Alastair Cameron, the Minister of State for Health, in pursuance of the powers conferred on me by subsection (1.) of section 134A of the *National Health Act* 1953-1955 hereby give notice that I have this day reprimanded Edward Maxwell Nicholls, of Mannum, medical practitioner, following investigation and report by the Medical Services Committee of Inquiry for the State of South Australia.

Dated this 6th day of June, 1956.

DONALD A. CAMERON,
Minister of State for Health.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales in accordance with the *Medical Practitioners Act*, 1938-1955:

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*a*) of the Act: Tassie, Gemmel Wilson, M.B., B.S., 1955 (Univ. Adelaide); Goodman, Joseph Victor, M.B., Ch.B., 1948 (Univ. New Zealand), D.M.R.D. (England), 1952, M.C.R.A., 1953; Waddell, John James, M.B., B.S., 1955 (Univ. Queensland).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*b*) of the Act: Nelson, Sarah, M.B., Ch.B., 1944 (Univ. St. Andrews), B.Sc. (Med.), 1941 (Univ. St. Andrews), D.C.H. (London), 1948, D.P.H. (London), 1955; Perkins, Michael Vivian, M.B., B.S., 1954 (Univ. London);

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JUNE 23, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2(1)	2(1)	4(3)	..	..	..	3	1	12
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1(1)	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3(1)	9(6)	2(1)	..	2	..	..	..	16
Diphtheria	1(1)	3(3)	2(1)	..	4(3)	..	..	..	10
Dysentery (Bacillary)	..	..	5(4)	1(1)	2	..	..	..	8
Encephalitis	..	..	1	1	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	63(34)	76(31)	..	5(5)	4(4)	5	1	1	155
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	6	..	..	..	..	..	6
Malaria	..	1(1)	..	..	..	..	4	..	5
Meningococcal Infection	..	1(1)	1	1(1)	1(1)	1(1)	..	..	5
Ophthalmia	..	..	..	..	..	..	1	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2	7(5)	2(2)	..	..	2	..	..	13
Puerperal Fever	..	..	1	..	..	1	..	..	2
Rubella	..	20(12)	..	4(1)	1(1)	..	..	..	25
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	21(12)	23(17)	7(2)	11(9)	1	1(1)	..	..	64
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	2(1)	1(1)	..	..	..	..	..	3
Trachoma	..	..	..	..	4	..	3	..	7
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	39(28)	27(22)	8(5)	1(1)	9(9)	5(4)	2	..	91
Typhoid Fever	1	..	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Egan, Michael Anthony, M.B., B.Ch., 1937 (N.U. Ireland); Summers, Frank, M.R.C.S., L.R.C.P., 1955 (London), M.B., B.Ch., 1955 (Univ. Cambridge); Cram, Morris, M.B., Ch.B., 1940 (Univ. Edinburgh).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (c) of the Act: Havas, Laszlo.

Registered medical practitioners who are required to complete twelve months' hospital service, pursuant to the provisions of Section 17 (3), and are registered under Section 17 (1) (a) of the Act: Fogland, William Gilbert, M.B., B.S., 1956 (Univ. Sydney).

Registered medical practitioners who are required to complete twelve months' hospital service, pursuant to the provisions of Section 17 (3), and are registered under Section 17 (1) (c) of the Act: Darvas, Frank Alexander.

The following additional qualifications have been registered: Cramer, John Earle, D.D.M. (Sydney), 1955; Furber, James Gordon, M.R.C.O.G. (England), 1956; Hobson, David Lennox, M.R.C.P. (London), 1956; Segal, Harry, M.S., 1948 (Univ. Sydney), F.R.C.S. (England), 1954; Tillett, John Varnell, D.M.R., R.C.P. and S., 1953, D.M.R. (London), 1953, M.C.R.A., 1953; Voss, John Henry Edward, M.R.A.C.P., 1955; Thwaite, Thomas John, D.L.O., R.C.P. and S., 1956; Hickie, John Bernard, M.R.C.P. (London), 1954, M.R.A.C.P., 1953; Craven, Richard Allan, F.R.A.C.S., 1956; Walters, Cecil Julian Manning, F.R.A.C.S., 1931; McDonald, Charles George, F.R.C.P. (London), 1956.

QUEENSLAND.

THE following have been registered, pursuant to the provisions of Section 19 (1) (a) and (d) of *The Medical Acts*, 1939 to 1955, of Queensland: Quinn, John Vincent, M.B., B.S., 1948 (Univ. Sydney); Paxton, Thomas Gordon, M.B., B.S., 1941 (Univ. London), M.R.C.P. (England), 1954; Gwynn, Arthur Montagu, M.B., Ch.B., 1942 (Univ. Aberdeen); Wright-Short, Frederick William, M.B., B.S., 1952 (Univ. Sydney); Waga, Witold Jerzy, M.B., Ch.B., 1942 (Polish School of Medicine, Edinburgh); Waga, Irene, M.B., Ch.B., 1948 (Polish School of Medicine, Edinburgh).

The following has been granted limited registration as a medical practitioner, pursuant to Section 20 (3) of *The Medical Acts*, 1939 to 1955, of Queensland: Bridger, Allan Joseph, M.B., B.S., 1956 (Univ. Sydney).

The following additional qualifications have been registered: Hood, Jessie Lurleen, D.M.R.T., R.C.P. (London), R.C.S. (England), 1955; Willson, Leslie, L.M. (Rotunda), 1955; Willson, Mary, D.C.H., R.C.P. and S. (Ireland), 1956.

Notice.

AUSTRALASIAN ASSOCIATION OF PSYCHIATRISTS (NEW SOUTH WALES BRANCH).

THE New South Wales Branch of the Australasian Association of Psychiatrists announces that a meeting for medical practitioners only will be held on Friday, August 17, 1956, at 8.30 p.m. at the Stawell Hall, 145 Macquarie Street, Sydney, on the subject of "Medical Aspects of Marriage Guidance". The speaker will be Professor David Mace, M.A., B.Sc., Ph.D., Professor of Human Relations at Drew University, Madison, New Jersey, United States of America. Professor Mace is making a lecture tour of Australia, which has been arranged by the National Marriage Guidance Council of Australia. He is one of the founders of the marriage guidance movement. All medical practitioners are invited to attend the meeting.

DEPARTMENT OF HEALTH, VICTORIA: SCHOOL MEDICAL SERVICE.

THE School Medical Service, Department of Health, Victoria, is organizing a four-day course in health education for school medical officers, to be held at "Stonnington", Glenferrie Road, Malvern, from September 3 to 6, 1956. A comprehensive programme has been arranged, and all school medical officers will be welcome. Details may be obtained from the Chief School Medical Inspector, 1 Somers Avenue, Malvern. As space is limited, early application to attend the course is advisable.

Deaths.

THE following deaths have been announced:

BYRNE.—Kevin Byrne, on July 3, 1956, at Sydney.

JAGERS.—Leslie James Jagers, on June 29, 1956, at Sydney.

CUSCADEN.—William George Henry Cuscaden, on July 1, 1956, at Lower Plenty, Victoria.

Medical Appointments.

Dr. A. W. Russell has been appointed Deputy Superintendent (Maternity Section) of the Queen Elizabeth Hospital, Adelaide.

Diary for the Month.

JULY 16.—Victorian Branch, B.M.A.: Finance Subcommittee.
JULY 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 18.—Western Australian Branch, B.M.A.: General Meeting.
JULY 19.—Victorian Branch, B.M.A.: Executive of Branch Council.
JULY 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 24.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

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